WEST Search History

DATE: Thursday, November 14, 2002

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=ADJ			
L5	L4 and restenosis	77	L5
L4	l2 same (polymorphism or mutation or variant)	745	L4
L3	L2 and restenosis	912	L3
L2	interleukin or IL-1$	15593	L2
L1	interleukin or IL$	216462	L1

END OF SEARCH HISTORY

Brain,disease...
stroke; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others
Interleukins...
[illegible]4; interleukin 1H4 and mature IL-1H4 for screening agonists and [illegible]ntagonists for treating inflammation, infection, cancer, autoimmune disease and others
CAS REGISTRY NUMBERS:
265295-30-5 343914-85-2 amino acid sequence; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others
175941-97-6 209511-54-6 224113-70-6 interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others
303170-58-3 nucleotide sequence; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others
343916-05-2 343916-06-3 unclaimed nucleotide sequence; interleukin-1 homolog, MAT IL-1H4
343779-79-3 unclaimed sequence; interleukin-1 homolog, MAT IL-1H4

4/7/13 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

134026093 CA: 134(3)26093t PATENT
Diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
INVENTOR(AUTHOR): Kornman, Kenneth S.; Duff, Gordon W.; Crossman, David C.; Francis, Sheila E.; Stephenson, Katherine
LOCATION: USA
ASSIGNEE: Interleukin Genetics, Inc.
PATENT: PCT International ; WO 200071753 A2 DATE: 20001130
APPLICATION: WO 2000US14299 (20000524) *US 317674 (19990524) *US 431352 (19991101)
PAGES: 129 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12Q-001/68A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZW; AT ; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA203003 Biochemical Genetics
CA201XXX Pharmacology
CA209XXX Biochemical Methods
CA214XXX Mammalian Pathological Biochemistry
CA215XXX Immunochemistry
IDENTIFIERS: interleukin 1 genotyping restenosis diagnosis therapeutic
DESCRIPTORS:
Nucleic acid hybridization...
allele-specific; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Artery,disease...

coronary, restenosis; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

Allele frequency... Anticoagulants... Antihypertensives... Antisense oligonucleotides... Anti-inflammatory agents... DNA sequence analysis... Drug screening... Genetic polymorphism... Genotyping(method)... Hypolipemic agents... Interleukin 1 receptor antagonist... Interleukin 1.alpha.... Interleukin 1.beta.... Interleukin 1... Mutation... Platelet aggregation inhibitors... Primers(nucleic acid)... RFLP(restriction fragment length polymorphism)... Ribozymes... SSCP(single-strand conformation polymorphism)... Test kits...

diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

Diagnosis...

genetic; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

Gene,animal...

IL-1RN; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

Gene,animal...

IL1A; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

Gene,animal...

IL1B; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

Heart,disease...

infarction; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

Blood vessel,disease...

occlusion; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

Genetic methods...

oligonucleotide ligation assay; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

Periodontium...

periodontitis; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

Nucleic acid amplification(method)...

primer-specific extension; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

Artery,disease...

restenosis; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

Oligonucleotides...

triple helix forming; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

CAS REGISTRY NUMBERS:

81295-04-7 81295-06-9 81295-40-1 84522-62-3 86352-30-9 92228-44-9 172306-44-4 diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

140744-82-7 140960-10-7 141002-08-6 nucleotide sequence; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

188135-75-3 188135-76-4 188135-77-5 188135-78-6 188135-79-7 193101-09-6 206073-47-4 224178-70-5 224178-77-2 249263-92-1 249263-94-3 249263-96-5 309983-89-9 309983-90-2 PCR primer; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

244295-37-2 244295-38-3 244295-41-8 244295-42-9 309983-91-3

309983-92-4 309983-93-5 309983-94-6 unclaimed nucleotide sequence; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

4/7/14 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

134014046 CA: 134(2)14046s PATENT
Diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
INVENTOR(AUTHOR): Francis, Sheila E.; Crossman, David C.; Duff, Gordon W.; Kornman, Kenneth S.
LOCATION: USA
ASSIGNEE: Interleukin Genetics, Inc.
PATENT: PCT International ; WO 200072015 A2 DATE: 20001130
APPLICATION: WO 2000US14775 (20000526) *US 320395 (19990526) *US 431352 (19991101)
PAGES: 122 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: G01N-033/53A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA203003 Biochemical Genetics
CA201XXX Pharmacology
CA209XXX Biochemical Methods
CA214XXX Mammalian Pathological Biochemistry
CA215XXX Immunochemistry
IDENTIFIERS: cardiovascular disease interleukin 1 gene family polymorphism, restenosis interleukin 1 gene family polymorphism, occlusive disorder interleukin 1 gene family polymorphism, fragile plaque disorder interleukin 1 gene family polymorphism, genotyping interleukin 1 gene family cardiovascular disease, RFLP interleukin 1 gene family cardiovascular disease
DESCRIPTORS:
Nucleic acid hybridization...
allele-specific; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Proteins,specific or class...
C-reactive, IL-1 genotypes correlated with; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Artery,disease...
coronary, restenosis; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
DNA sequence analysis... Drug screening... Genotyping(method)... Interleukin 1 receptor antagonist... Interleukin 1.alpha.... Interleukin 1.beta.... Interleukin 1... Mutation... Primers(nucleic acid)... RFLP(restriction fragment length polymorphism)... SSCP(single-strand conformation polymorphism)... Susceptibility(genetic)... Test kits...
diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family

Cardiovascular system...
disease; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Blood vessel,disease...
fragile plaque; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Diagnosis...
genetic; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Gene,animal...
IL-1RN; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Gene,animal...
IL1A; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Gene,animal...
IL1B; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Heart,disease...
infarction; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Lipoproteins...
low-d., IL-1 genotypes correlated with; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Lipoproteins...
Lp(a), IL-1 genotypes correlated with; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Angiogenesis...
neovascularization, IL-1 genotypes correlated with; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Blood vessel,disease...
occlusion; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Genetic methods...
oligonucleotide ligation assay; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Periodontium...
periodontitis; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Nucleic acid amplification(method)...
primer-specific extension; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Artery,disease...
restenosis; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Brain,disease...
stroke; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family

CAS REGISTRY NUMBERS:

57-88-5 biological studies, IL-1 genotypes correlated with; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
81295-04-7 81295-06-9 81295-40-1 81811-56-5 84522-62-3 86352-30-9

92228-44-9 diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
188135-76-4 188135-77-5 188135-78-6 188135-79-7 193101-09-6 193101-10-9 224178-77-2 249263-92-1 249263-94-3 249263-96-5 PCR primer; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
244295-37-2 244295-38-3 244295-41-8 244295-42-9 309983-91-3 309983-92-4 309983-93-5 309983-94-6 unclaimed nucleotide sequence; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family

4/7/15 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

133262303 CA: 133(19)262303m PATENT
Human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
INVENTOR(AUTHOR): Hayden, Michael R.; Wilson, Angela R.; Pimstone, Simon N.
LOCATION: Can.,
ASSIGNEE: University of British Columbia; Xenon Bioresearch, Inc.
PATENT: PCT International ; WO 200055318 A2 DATE: 20000921
APPLICATION: WO 2000IB532 (20000315) *US PV124702 (19990315) *US PV138048 (19990608) *US PV139600 (19990617) *US PV151977 (19990901)
PAGES: 229 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-015/12A; C07K-014/705B; C12N-005/10B; A01K-067/027B; C12N-015/00B; A61K-038/17B; A61K-048/00B; A61K-038/45B; A61K-031/00B; A61K-031/70B; G01N-033/68B; C12Q-001/68B; C12N-015/11B DESIGNATED COUNTRIES: AE; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SL; SZ ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA203003 Biochemical Genetics
CA201XXX Pharmacology
CA206XXX General Biochemistry
CA213XXX Mammalian Biochemistry
IDENTIFIERS: sequence human gene ABC1 transporter cDNA, cholesterol transport familial HDL deficiency ABC1 gene mutation
DESCRIPTORS:
Transport proteins...
ABC1 (ATP binding cassette-contg. 1); human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Genetic vectors...
ABC1 gene-contg.; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
RNA splicing...
ABC1 mutation affecting, disease and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Interleukin 1.beta....
ABC1 transport of; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Gene,animal...

ABC1; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Chicken(Gallus domesticus)... Mammal(Mammalia)...
ABC1/ABC1 mutant-expressing; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Brain,disease...
cerebrovascular; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Artery,disease...
coronary, restenosis; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Artery,disease...
coronary; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Mutation...
deletion, of ABC1 gene, disease and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Cardiovascular system...
disease; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Disease,animal...
familial HDL deficiency, ABC1 gene mutations and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

cDNA sequences...
for human ABC1 transporter

Mutation...
frameshift, of ABC1 gene, disease and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Lipoproteins...
high-d., cholesterol; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Alzheimer's disease... Neoplasm... Niemann-Pick disease...
human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Chromosome...
human 9, ABC1 gene mapped to; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Nervous system...
Huntington's chorea; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Mutation...
nonsense, of ABC1 gene, disease and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Genetic mapping... Genetic polymorphism... mRNA... Promoter(genetic element)... RFLP(restriction fragment length polymorphism)...
of ABC1 gene; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Biological transport...
of cholesterol; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Protein sequences...
of human ABC1 transporter

DNA sequences...
of human ABC1 transporter gene ABC1 exons and flanks

Blood vessel,disease...
peripheral; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Mutation...
substitution, of ABC1 gene, disease and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Disease,animal...
Tangier, ABC1 gene mutations and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Antidiabetic agents...
thiozolidinediones, modulation of ABC1 with; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Brain,disease...
X-linked adrenoleukodystrophy; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

CAS REGISTRY NUMBERS:

296343-83-4 296343-89-0 296787-98-9 amino acid sequence; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
56-65-5 biological studies, binding/hydrolysis by ABC1 of; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
57-88-5 biological studies, human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
50-28-2 59-67-6 biological studies, modulation of ABC1 with; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
943-45-3D derivs., inhibitors, modulation of ABC1 with; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
506-32-1D derivs., modulation of ABC1 with; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
9028-35-7 71160-24-2 inhibitors, modulation of ABC1 with; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
18104-45-5 28822-58-4 37353-31-4 50892-23-4 78111-17-8 98462-03-4 98524-19-7 141436-78-4 142008-29-5 modulation of ABC1 with; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
80449-06-5 81295-19-4 116155-80-7 131754-84-2 mutation affecting ABC1 cleavage by; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
233742-58-0 233742-64-8 296343-69-6 296343-70-9 296343-71-0 296343-72-1 296343-73-2 296343-74-3 296343-75-4 296343-76-5 296343-77-6 296343-80-1 296343-81-2 296343-82-3 296343-84-5 296343-85-6 296343-86-7 296343-87-8 296343-88-9 296343-90-3 296363-75-2 nucleotide sequence; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
296354-50-2 296354-51-3 296354-52-4 296354-53-5 296354-54-6 296354-55-7 296354-56-8 296354-57-9 296354-58-0 296354-59-1 296354-60-4 296354-61-5 296354-62-6 296354-63-7 296354-64-8 296354-65-9 296354-66-0 296354-67-1 296354-68-2 296354-69-3 296354-70-6 296354-71-7 296354-72-8 296354-73-9 296354-74-0 296354-75-1 296354-76-2 296354-77-3 296354-78-4 296354-79-5 296354-80-8 296354-81-9 296354-82-0 296354-83-1 296354-84-2 296354-85-3 296354-86-4 296354-87-5 296354-88-6 296354-89-7 296354-90-0 296354-91-1 296354-92-2 296354-93-3 296354-94-4 296354-95-5 296354-96-6 296354-97-7 296354-98-8 296354-99-9 296355-00-5 296355-01-6 296355-02-7 296355-03-8 296355-04-9 296355-05-0 296355-06-1 296355-07-2 296355-08-3 296355-09-4

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    296356-85-9  296356-86-0  296356-87-1  296356-88-2  296356-89-3
    296356-90-6  296356-91-7  unclaimed nucleotide sequence; human ABC1
    transporter and DNA and methods for modulating cholesterol levels and
    diagnosing disease
296237-28-0  296237-29-1  296237-30-4  296237-31-5  296237-32-6
    296237-33-7  296237-34-8  296237-35-9  296237-36-0  296237-37-1
    296237-38-2  296237-39-3  296237-40-6  296237-41-7  296237-42-8
    296237-43-9  unclaimed sequence; human ABC1 transporter and DNA and
    methods for modulating cholesterol levels and diagnosing disease
?
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!


? <------User Break------>

>>>Unrecognizable Command
? s l2 and (ACE or angiotensin or angiotensinogen)

          34750  L2
          76049  ACE
         342370  ANGIOTENSIN
          13483  ANGIOTENSINOGEN
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      S5       38  L2 AND (ACE OR ANGIOTENSIN OR ANGIOTENSINOGEN)
? s s2 and (ace or angiotensin or angiotensinogen)

            584  S2
          76049  ACE
         342370  ANGIOTENSIN
          13483  ANGIOTENSINOGEN
      S6     260  S2 AND (ACE OR ANGIOTENSIN OR ANGIOTENSINOGEN)
? rd s6

...examined 50 records  (50)
...examined 50 records  (100)
...examined 50 records  (150)
...examined 50 records  (200)
...examined 50 records  (250)
...completed examining records
      S7     116  RD S6 (unique items)
? t s7/6/1-116


 7/6/1      (Item 1 from file: 5)
13066485   BIOSIS NO.: 200100273634
Effect of ACE inhibitors on angiographic restenosis after
  coronary stenting (PARIS): A randomised, double-blind, placebo-controlled
  trial.
2001


 7/6/2      (Item 2 from file: 5)
12994678   BIOSIS NO.: 200100201827
ACE D/I polymorphism and incidence of post-PTCA
  restenosis: A prospective, angiography-based evaluation.
2001


 7/6/3      (Item 3 from file: 5)
12970606   BIOSIS NO.: 200100177755
Angiotensin converting enzyme DD genotype affects the changes of
  plasma plasminogen activator inhibitor-1 activity after primary
  percutaneous transluminal coronary angioplasty in acute myocardial
  infarction patients.
2000


 7/6/4      (Item 4 from file: 5)
12947456   BIOSIS NO.: 200100154605
Angiotensin-converting enzyme insertion/deletion polymorphism
  and restenosis after coronary stenting: A meta-analysis.
2001


 7/6/5      (Item 5 from file: 5)
12947438   BIOSIS NO.: 200100154587
Insertion/deletion polymorphism of angiotensin I-converting
  enzyme gene and risk of restenosis after directional coronary
  atherectomy followed by stent implantation.
2001
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7/6/6 (Item 6 from file: 5)
12763686 BIOSIS NO.: 200000517309
The insertion/deletion **polymorphism** of the **angiotensin** I converting enzyme is not associated with in-stent **restenosis** in diabetic patients.
2000

7/6/7 (Item 7 from file: 5)
12763672 BIOSIS NO.: 200000517295
Insertion/deletion **polymorphism** of **angiotensin**-converting enzyme gene and risk of **restenosis** after directional coronary atherectomy followed by stent implantation.
2000

7/6/8 (Item 8 from file: 5)
12642892 BIOSIS NO.: 200000396394
The **angiotensinogen** gene 235T variant is associated with an increased risk of **restenosis** after percutaneous transluminal coronary angioplasty.
2000

7/6/9 (Item 9 from file: 5)
12620272 BIOSIS NO.: 200000373774
Insertion/deletion **polymorphism** of the **angiotensin** I-converting enzyme gene is not associated with **restenosis** after coronary stent placement.
2000

7/6/10 (Item 10 from file: 5)
12554135 BIOSIS NO.: 200000307637
Role of conventional risk factors and the A/C **polymorphism** of the **angiotensin**-II type 1 receptor gene in post-menopausal women treated with coronary stenting.
2000

7/6/11 (Item 11 from file: 5)
12554134 BIOSIS NO.: 200000307636
I/D **polymorphism** of the **ACE** gene as a marker of different cardiovascular diseases. Relations with hypertension, coronary atherosclerosis and **restenosis** after coronary stent implantion.
2000

7/6/12 (Item 12 from file: 5)
12403842 BIOSIS NO.: 200000157344
Polymorphisms of the **angiotensin** converting enzyme and the **angiotensin** II Type 1 receptor genes in in-stent **restenosis**. Is there a synergism?
2000

7/6/13 (Item 13 from file: 5)

12403841 BIOSIS NO.: 200000157343
Angiotensin converting enzyme gene **polymorphism** and **restenosis** rate after coronary stenting.
2000

7/6/14 (Item 14 from file: 5)
12284411 BIOSIS NO.: 200000042278
The **angiotensin** converting enzyme intron 16 **polymorphism** in patients with hereditary venous thromboembolism.
1999

7/6/15 (Item 15 from file: 5)
12281187 BIOSIS NO.: 200000034689
Genetic risk factors for post-PTCA **restenosis**: A comprehensive analysis of multiple candidate genes.
1999

7/6/16 (Item 16 from file: 5)
12268762 BIOSIS NO.: 200000022264
The effect of quinapril on prevention of **restenosis** after coronary stenting were influenced by the insertion/deletion **polymorphism** in the **angiotensin** I-converting enzyme gene.
1999

7/6/17 (Item 17 from file: 5)
12256651 BIOSIS NO.: 200000010153
Pharmacogenetic analysis of the effect of **angiotensin**-converting enzyme inhibitor on **restenosis** after percutaneous transluminal coronary angioplasty.
1999

7/6/18 (Item 18 from file: 5)
12219937 BIOSIS NO.: 199900514786
Insertion/deletion **polymorphism** of the **angiotensin**-converting enzyme and **restenosis** after coronary stent placement.
1998

7/6/19 (Item 19 from file: 5)
12211510 BIOSIS NO.: 199900506359
Not the **ACE** gene **polymorphism**, but the E-selectin gene **polymorphism** helps to predict **restenosis** after coronary angioplasty.
1998

7/6/20 (Item 20 from file: 5)
12199877 BIOSIS NO.: 199900494726
Impact of conventional risk factors and the **angiotensin**-converting enzyme in women treated with coronary stent placement.
1999

7/6/21 (Item 21 from file: 5)
12192450 BIOSIS NO.: 199900487299
Insertion/deletion **polymorphism** of the **angiotensin** I-converting enzyme gene and 1-year angiographic and clinical outcome after coronary stent placement.
1999

7/6/22 (Item 22 from file: 5)
12192449 BIOSIS NO.: 199900487298
Long-term clinical outcome of coronary stenting: Role of the D **allele** of the **ACE** gene, and importance of the angiographic pattern of **restenosis**.
1999

7/6/23 (Item 23 from file: 5)
12192446 BIOSIS NO.: 199900487295
The PI A1/A2 **polymorphism** of the platelet glycoprotein IIIa is not associated with coronary stent **restenosis**.
1999

7/6/24 (Item 24 from file: 5)
12175417 BIOSIS NO.: 199900470266
Angiotensin II receptor subtype gene expression after stent implantation in a pig model.
1999

7/6/25 (Item 25 from file: 5)
11743780 BIOSIS NO.: 199800524476
Effect of **angiotensin**-converting enzyme inhibitor in preventing **restenosis** after percutaneous transluminal coronary angioplasty.
1998

7/6/26 (Item 26 from file: 5)
11743115 BIOSIS NO.: 199800523811
Polymorphism of **angiotensin** I-converting enzyme and risk coronary stent **restenosis**.
1998

7/6/27 (Item 27 from file: 5)
11743113 BIOSIS NO.: 199800523809
The deletion **allele** of insertion/deletion **polymorphism** of the **angiotensin**-converting enzyme gene: A real marker of genetic predisposition for stent **restenosis**.
1998

7/6/28 (Item 28 from file: 5)
11741680 BIOSIS NO.: 199800522376
Significance of **angiotensin** I-converting enzyme gene **polymorphism** as risk factor for stent **restenosis**.
1998

7/6/29 (Item 29 from file: 5)
11710520 BIOSIS NO.: 199800492251
Association between the **ACE** genotype and coronary artery disease: Insights from studies on **restenosis**, vasomotion and thrombosis.
1998

7/6/30 (Item 30 from file: 5)
11368199 BIOSIS NO.: 199800149531
The D **allele** of the **angiotensin** I converting enzyme is associated with diffuse in-stent **restenosis**.
1998

7/6/31 (Item 31 from file: 5)
11330428 BIOSIS NO.: 199800111760
Insertion/deletion **polymorphism** and plasma levels of **Angiotensin** I Converting Enzyme in coronary stent **restenosis**.
1997

7/6/32 (Item 32 from file: 5)
11308348 BIOSIS NO.: 199800089680
Dual determination of **angiotensin**-converting enzyme and **angiotensin**-II type 1 receptor genotypes as predictors of **restenosis** after coronary angioplasty.
1998

7/6/33 (Item 33 from file: 5)
11308302 BIOSIS NO.: 199800089634
Plasma activity and insertion/deletion **polymorphism** of **angiotensin** I-converting enzyme: A major risk factor and a marker of risk for coronary stent **restenosis**.
1998

7/6/34 (Item 34 from file: 5)
11237775 BIOSIS NO.: 199800019107
The I/D **polymorphism** of the **angiotensin** converting enzyme gene and in-stent **restenosis** in diabetic and non-diabetic patients.
1997

7/6/35 (Item 35 from file: 5)
11237451 BIOSIS NO.: 199800018783
Angiotensin-converting enzyme insertion/deletion **polymorphism** and **restenosis** after directional coronary atherectomy.
1997

7/6/36 (Item 36 from file: 5)
11234326 BIOSIS NO.: 199800015658
The pattern (focal/diffuse) of angiographic in-stent **restenosis** is associated to the I/D **polymorphism** of the **angiotensin** converting enzyme gene.

1997

7/6/37 (Item 37 from file: 5)
11216629 BIOSIS NO.: 199799837774
Gene **polymorphism** of **angiotensin** II type 1 receptor and **angiotensinogen** in patients with coronary artery disease.
1997

7/6/38 (Item 38 from file: 5)
11216563 BIOSIS NO.: 199799837708
Angiotensin-converting enzyme insertion/deletion **polymorphism** and **restenosis** after coronary intervention.
1997

7/6/39 (Item 39 from file: 5)
11175614 BIOSIS NO.: 199799796759
Renin-**angiotensin** system: Genes to beside.
1997

7/6/40 (Item 40 from file: 5)
11158418 BIOSIS NO.: 199799779563
Plasma **angiotensin**-converting enzyme and D/D genotype of the **ACE** gene: Telltales and deceptions.
1997

7/6/41 (Item 41 from file: 5)
11157587 BIOSIS NO.: 199799778732
Influence of the **angiotensin**-converting-enzyme gene **polymorphism** on the **restenosis** rate after interventional therapy of coronary artery disease.
1997

7/6/42 (Item 42 from file: 5)
11021825 BIOSIS NO.: 199799642970
D **allele** of the **angiotensin** I-converting enzyme is a major risk factor for **restenosis** after coronary stenting.
1997

7/6/43 (Item 43 from file: 5)
10893077 BIOSIS NO.: 199799514222
The expression of **angiotensin**-I converting enzyme in human atherosclerotic plaques is not related to the deletion/insertion **polymorphism** but to the risk of **restenosis** after coronary interventions.
1997

7/6/44 (Item 44 from file: 5)
10825937 BIOSIS NO.: 199799447082
The absence of D **allele** of the **ACE** ID genotype prevents **restenosis** after coronary stenting: A quantitative angiographic

study.
1997

7/6/45 (Item 45 from file: 5)
10660992 BIOSIS NO.: 199699282137
Plasma **angiotensin**-converting enzyme levels, and insertion/deletion **polymorphism** of the enzyme, as predictors of **restenosis** after elective coronary stenting.
1996

7/6/46 (Item 46 from file: 5)
10525532 BIOSIS NO.: 199699146677
Apolipoprotein E **polymorphism** does not predict risk of **restenosis** after coronary angioplasty.
1996

7/6/47 (Item 47 from file: 5)
10413859 BIOSIS NO.: 199699035004
Angiotensin-converting enzyme insertion/deletion **polymorphism** and **restenosis** after coronary angioplasty in unstable angina pectoris.
1996

7/6/48 (Item 48 from file: 5)
10126875 BIOSIS NO.: 199698581793
Angiotensin converting enzyme and apolipoprotein E genotypes interact in **restenosis** after coronary angioplasty.
1995

7/6/49 (Item 49 from file: 5)
10126712 BIOSIS NO.: 199698581630
Relation between the deletion **polymorphism** of the **angiotensin** -converting enzyme gene and late luminal narrowing after coronary angioplasty.
1995

7/6/50 (Item 50 from file: 5)
10126656 BIOSIS NO.: 199698581574
Polymorphism in the **angiotensin** I-converting enzyme and apolipoprotein E genes and **restenosis** after coronary angioplasty in men.
1995

7/6/51 (Item 51 from file: 5)
10088856 BIOSIS NO.: 199598543774
Angiotensin-converting enzyme and apolipoprotein E genotypes and **restenosis** after coronary angioplasty.
1995

7/6/52 (Item 52 from file: 5)

09916666 BIOSIS NO.: 199598371584
Angiotensin-I converting enzyme genotype DD is a risk factor for coronary artery disease.
1995

7/6/53 (Item 53 from file: 5)
09904591 BIOSIS NO.: 199598359509
Angiotensin-converting enzyme gene **polymorphism** and **restenosis** after PTCA.
1995

7/6/54 (Item 54 from file: 5)
09854362 BIOSIS NO.: 199598309280
Enhanced predictability of myocardial infarction in Japanese by combined genotype analysis.
1995

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09808685 BIOSIS NO.: 199598263603
Insertion/deletion **polymorphism** in the **angiotensin**-converting enzyme gene and risk of **restenosis** after coronary angioplasty.
1995

7/6/56 (Item 56 from file: 5)
09775952 BIOSIS NO.: 199598230870
Molecular variant of **angiotensinogen** gene is associated with coronary atherosclerosis.
1995

7/6/57 (Item 57 from file: 5)
09704462 BIOSIS NO.: 199598159380
Does Insertion/Deletion **Polymorphism** of the **Angiotensin** Converting Enzyme Influence the Occurrence of **Restenosis** After Percutaneous Transluminal Coronary Angioplasty?
1995

7/6/58 (Item 58 from file: 5)
09633694 BIOSIS NO.: 199598088612
DD Genotype of the **Angiotensin**-Converting Enzyme Gene Is a Risk Factor for Left Ventricular Hypertrophy.
1994

7/6/59 (Item 59 from file: 5)
09567015 BIOSIS NO.: 199598021933
Angiotensin converting enzyme genotype DD is a potent risk factor for coronary artery disease and **restenosis** post percutaneous transluminal angioplasty.
1994

7/6/60 (Item 60 from file: 5)

09450307 BIOSIS NO.: 199497458677
Cell biology and genetics of **angiotensin** in cardiovascular disease.
1994

7/6/61 (Item 1 from file: 34)
09901385 Genuine Article#: 464BK Number of References: 16
Title: Is there a role of **angiotensin**-converting enzyme gene **polymorphism** in the failure of arteriovenous femoral shunts for hemodialysis? (ABSTRACT AVAILABLE)
Publication date: 20010700

7/6/62 (Item 2 from file: 34)
09493313 Genuine Article#: 413XJ Number of References: 31
Title: Ribozyme-mediated inhibition of rat leukocyte-type 12-lipoxygenase prevents intimal hyperplasia in balloon-injured rat carotid arteries (ABSTRACT AVAILABLE)
Publication date: 20010313

7/6/63 (Item 3 from file: 34)
09481152 Genuine Article#: 408KN Number of References: 56
Title: Polymorphisms of the renin-**angiotensin** system in patients with multifocal renal arterial fibromuscular dysplasia (ABSTRACT AVAILABLE)
Publication date: 20010300

7/6/64 (Item 4 from file: 34)
09433771 Genuine Article#: 405VU Number of References: 32
Title: Endothelium-independent conversion of **angiotensin** I by vascular smooth muscle cells (ABSTRACT AVAILABLE)
Publication date: 20010200

7/6/65 (Item 5 from file: 34)
09259934 Genuine Article#: 384FE Number of References: 39
Title: **ACE**-gene **polymorphism** is associated with the development of allograft vascular disease in heart transplant recipients (ABSTRACT AVAILABLE)
Publication date: 20001200

7/6/66 (Item 6 from file: 34)
09259141 Genuine Article#: 385PR Number of References: 20
Title: Relation between the insertion/deletion **polymorphism** of the **angiotensin** I converting enzyme gene and **restenosis** after coronary stenting (ABSTRACT AVAILABLE)
Publication date: 20001200

7/6/67 (Item 7 from file: 34)
09014018 Genuine Article#: 356MU Number of References: 18
Title: Relationship between plasma **ACE** activity and the proliferative healing process in coronary vessel injury after coronary stenting
Publication date: 20000900

7/6/68 (Item 8 from file: 34)
08537163 Genuine Article#: 297RX Number of References: 24
Title: Searching for a better assessment of the individual coronary risk profile - The role of **angiotensin**-converting enzyme, **angiotensin** II type 1 receptor and **angiotensinogen** gene polymorphisms (ABSTRACT AVAILABLE)
Publication date: 20000400

7/6/69 (Item 9 from file: 34)
08503639 Genuine Article#: 293UK Number of References: 125
Title: Genetic risk factors and **restenosis** after percutaneous coronary interventions (ABSTRACT AVAILABLE)
Publication date: 20000200

7/6/70 (Item 10 from file: 34)
08503636 Genuine Article#: 293UK Number of References: 77
Title: Genetic risk faktors for myocardial infarction (ABSTRACT AVAILABLE)
Publication date: 20000200

7/6/71 (Item 11 from file: 34)
08068159 Genuine Article#: 233CM Number of References: 0
Title: **Angiotensin** converting enzyme (I/D) **polymorphism** (**ACE**-I/D), apolipoprotein E (ApoE) genotype and **restenosis** after peripheral angioplasty.
Publication date: 19990700

7/6/72 (Item 12 from file: 34)
07846756 Genuine Article#: 215LK Number of References: 10
Title: Coronary stent implantation and **restenosis**. Can be predicted by examining the genes?
Publication date: 19990700

7/6/73 (Item 13 from file: 34)
07846755 Genuine Article#: 215LK Number of References: 20
Title: D/D **polymorphism** of the **ace** gene might be a risk factor for in-stent **restenosis** (ABSTRACT AVAILABLE)
Publication date: 19990700

7/6/74 (Item 14 from file: 34)
07174531 Genuine Article#: 131TZ Number of References: 0
Title: The 235T **allele** of the **angiotensinogen** gene and the risk for **restenosis** after first and second coronary angioplasty
Publication date: 19981000

7/6/75 (Item 15 from file: 34)
06512314 Genuine Article#: YY084 Number of References: 50
Title: Donor **ACE** gene **polymorphism**: a genetic risk factor for accelerated coronary sclerosis following cardiac transplantation (ABSTRACT AVAILABLE)
Publication date: 19980200

7/6/76 (Item 16 from file: 34)
06169254 Genuine Article#: XZ516 Number of References: 81
Title: Renin-**angiotensin** system: Genes to bedside (ABSTRACT AVAILABLE)
Publication date: 19970900

7/6/77 (Item 17 from file: 34)
06053694 Genuine Article#: XE898 Number of References: 0
Title: **Angiotensin**-converting enzyme gene **polymorphism** and risk of **restenosis** after coronary stenting
Publication date: 19970600

7/6/78 (Item 18 from file: 34)
05912621 Genuine Article#: XF079 Number of References: 20
Title: Bail-out stent implantation of a muscle bridging in the left anterior descending artery after post-interventional dissection (ABSTRACT AVAILABLE)
Publication date: 19970500

7/6/79 (Item 19 from file: 34)
05821778 Genuine Article#: XA009 Number of References: 81
Title: Hypothesis: An **angiotensin** converting enzyme genotype, present in one in three Caucasians, is associated with an increased mortality rate (ABSTRACT AVAILABLE)
Publication date: 19960100

7/6/80 (Item 20 from file: 34)
05813166 Genuine Article#: WZ337 Number of References: 37
Title: Impact of molecular biology on cardiovascular medicine (ABSTRACT AVAILABLE)
Publication date: 19970400

7/6/81 (Item 21 from file: 34)
05730281 Genuine Article#: WT791 Number of References: 10
Title: High incidence of **angiotensin** I converting enzyme genotype II in Kawasaki disease patients with coronary aneurysm (ABSTRACT AVAILABLE)
Publication date: 19970400

7/6/82 (Item 22 from file: 34)
05615487 Genuine Article#: WK890 Number of References: 31
Title: Non-invasive coronary angiography by electron beam tomography: Methods and clinical evaluation in the follow-up after PTCA (ABSTRACT AVAILABLE)
Publication date: 19970200

7/6/83 (Item 23 from file: 34)
05610641 Genuine Article#: WK743 Number of References: 206
Title: Toward molecular strategies for heart disease - Past, present, future (ABSTRACT AVAILABLE)

Publication date: 19970200

7/6/84 (Item 24 from file: 34)
05390275 Genuine Article#: VV335 Number of References: 87
Title: WHAT CAN **ANGIOTENSIN** ANTAGONISTS DO THAT CONVERTING-ENZYME INHIBITION CANT - THE CASE OF POST-ANGIOPLASTIC **RESTENOSIS** (Abstract Available)

7/6/85 (Item 25 from file: 34)
05210660 Genuine Article#: VH619 Number of References: 6
Title: **ACE POLYMORPHISM,** A GENETIC PREDICTOR OF OCCLUSION AFTER CORONARY ANGIOPLASTY

7/6/86 (Item 26 from file: 34)
05069873 Genuine Article#: TK257 Number of References: 23
Title: A VARIANT OF HUMAN PARAOXONASE ARYLESTERASE (HUMPONA) GENE IS A RISK FACTOR FOR CORONARY-ARTERY DISEASE (Abstract Available)

7/6/87 (Item 27 from file: 34)
05019997 Genuine Article#: UZ529 Number of References: 43
Title: INSERTION/DELETION **POLYMORPHISM** IN THE **ANGIOTENSIN** -CONVERTING ENZYME GENE AND RISK OF AND PROGNOSIS AFTER MYOCARDIAL-INFARCTION (Abstract Available)

7/6/88 (Item 28 from file: 34)
04964043 Genuine Article#: UV871 Number of References: 166
Title: THROMBOSIS IN ISCHEMIC-HEART-DISEASE (Abstract Available)

7/6/89 (Item 29 from file: 34)
04798958 Genuine Article#: UH635 Number of References: 135
Title: **ANGIOTENSIN** RECEPTORS AND THEIR THERAPEUTIC IMPLICATIONS (Abstract Available)

7/6/90 (Item 30 from file: 34)
03966914 Genuine Article#: QW013 Number of References: 0
Title: THE MOLECULAR AND CELLULAR BIOLOGY OF HEART-FAILURE (Abstract Available)

7/6/91 (Item 31 from file: 34)
03747452 Genuine Article#: QC653 Number of References: 71
Title: PHARMACOKINETICS OF ADENOVIRAL VECTOR-MEDIATED GENE DELIVERY TO VASCULAR SMOOTH-MUSCLE CELLS - MODULATION BY POLOXAMER-407 AND IMPLICATIONS FOR CARDIOVASCULAR GENE-THERAPY (Abstract Available)

7/6/92 (Item 32 from file: 34)
03487243 Genuine Article#: PH234 Number of References: 30
Title: EFFECTS OF **ACE**-INHIBITORS ON CORONARY ATHEROSCLEROSIS AND **RESTENOSIS**

7/6/93 (Item 33 from file: 34)
03367612 Genuine Article#: PA636 Number of References: 4
Title: ARE POLYMORPHISMS IN THE **ACE** GENE A POTENT GENETIC RISK FACTOR FOR **RESTENOSIS**

7/6/94 (Item 34 from file: 34)
02871146 Genuine Article#: ML145 Number of References: 5
Title: A POTENT GENETIC RISK FACTOR FOR **RESTENOSIS**

7/6/95 (Item 35 from file: 34)
02869323 Genuine Article#: ML149 Number of References: 251
Title: THE TRANSITION FROM EXPERIMENTAL TO CLINICAL-PHARMACOLOGY - THE RENIN-**ANGIOTENSIN** PARADIGM (Abstract Available)

7/6/96 (Item 36 from file: 34)
02799510 Genuine Article#: ME801 Number of References: 54
Title: RENIN-**ANGIOTENSIN** SYSTEM GENES AS CANDIDATE GENES IN CARDIOVASCULAR-DISEASES (Abstract Available)

7/6/97 (Item 37 from file: 34)
02799164 Genuine Article#: ME822 Number of References: 9
Title: **ANGIOTENSIN**-CONVERTING ENZYME **POLYMORPHISM** IN HYPERTROPHIC CARDIOMYOPATHY AND SUDDEN CARDIAC DEATH (Abstract Available)

7/6/98 (Item 38 from file: 34)
02799161 Genuine Article#: ME822 Number of References: 13
Title: **ANGIOTENSIN**-CONVERTING ENZYME-DD GENOTYPE IN PATIENTS WITH ISCHEMIC OR IDIOPATHIC DILATED CARDIOMYOPATHY (Abstract Available)

7/6/99 (Item 39 from file: 34)
02455203 Genuine Article#: LC377 Number of References: 48
Title: INTRARENAL LOCALIZATION OF **ANGIOTENSIN**-II TYPE-1 RECEPTOR MESSENGER-RNA IN THE RAT (Abstract Available)

7/6/100 (Item 40 from file: 34)
02387016 Genuine Article#: KY052 Number of References: 48
Title: DIFFERENTIAL-EFFECTS OF RENIN-**ANGIOTENSIN** SYSTEM BLOCKADE ON ATHEROGENESIS IN CHOLESTEROL-FED RABBITS (Abstract Available)

7/6/101 (Item 1 from file: 65)
02926947 INSIDE CONFERENCE ITEM ID: CN030875551
Coronary Stent **Restenosis**: Insertion/deletion **Polymorphism** and Plasma Levels of **Angiotensin** I Converting Enzyme
CONFERENCE: Associazione Genetica Italiana-Convegno scientifico; 43 (199709)

7/6/102 (Item 1 from file: 73)

10727690 EMBASE No: 2000137046
The insertion/deletion **polymorphism** of the **angiotensin**
-converting enzyme gene and the risk for **restenosis** after PTCA
2000

7/6/103 (Item 2 from file: 73)
07401143 EMBASE No: 1998313401
Association between the **ACE** genotype and coronary artery disease
1998

7/6/104 (Item 3 from file: 73)
06928427 EMBASE No: 1997212906
Genetics of interventional cardiology: Old principles, new frontiers
1997

7/6/105 (Item 4 from file: 73)
06568306 EMBASE No: 1996229855
Fibrinolysis and risk of coronary artery disease
1996

7/6/106 (Item 5 from file: 73)
06372788 EMBASE No: 1996036519
Angiotensin I converting enzyme gene **polymorphism** and
coronary heart disease
1995

7/6/107 (Item 1 from file: 94)
04348252 JICST ACCESSION NUMBER: 99A0746604 FILE SEGMENT: JICST-E
Angiotensin II type I receptor and **angiotensinogen** gene
polymorphisms in patients with coronary artery disease., 1999

7/6/108 (Item 2 from file: 94)
03563160 JICST ACCESSION NUMBER: 98A0404211 FILE SEGMENT: JICST-E
Deletion **polymorphism** of **angiotensin** I-converting enzyme gene
associates with increased risk for acute myocardial infarction., 1998

7/6/109 (Item 1 from file: 98)
03766798 H.W. WILSON RECORD NUMBER: BGSI98016798 (USE FORMAT 7 FOR
FULLTEXT)
The physiology of parathyroid hormone-related protein: an emerging role as
a developmental factor.
WORD COUNT: 12247
'98 (19980000)

7/6/110 (Item 2 from file: 98)
03546695 H.W. WILSON RECORD NUMBER: BGSI97046695 (USE FORMAT 7 FOR
FULLTEXT)
Cellular and molecular mechanisms of pulmonary vascular remodeling.
WORD COUNT: 25314
'97 (19970000)

7/6/111 (Item 3 from file: 98)
03536366 H.W. WILSON RECORD NUMBER: BGSI97036366
Genetic make-up predicts success of cornary stenting.
July 5 '97 (19970705)

7/6/112 (Item 1 from file: 144)
14406734 PASCAL No.: 00-0062501
ACE gene **polymorphism** and coronary **restenosis**
Biological determinants of atherosclerosis and **restenosis**
1999

7/6/113 (Item 2 from file: 144)
13475066 PASCAL No.: 98-0172121
EPIDEMIOLOGIE DE LA MALADIE CORONAIRE : RECHERCHE DES DETERMINANTS GENETIQUES DE LA VASOMOTRICITE CORONAIRE ET DE LA RESTENOSE
(CORONARY DISEASE EPIDEMIOLOGY : CORONARY AND **RESTENOSIS** GENETIC RISK FACTORS DETERMINATION) 1997-12; 1997

7/6/114 (Item 1 from file: 155)
10495287 20135076 PMID: 10670180
I/D **polymorphism** of the **ACE** gene and A1166C of the AT1R gene as risk factors for **restenosis** after coronary angioplasty]
Polimorfismo I/D del gene **ACE** e A1166C del gene AT1R quali fattori di rischio di restenosi dopo angioplastica coronarica.
Dec 1999

7/6/115 (Item 2 from file: 155)
10257485 99368525 PMID: 10439670
D/D genotype of the gene for **angiotensin** converting enzyme as a risk factor for post-stent coronary **restenosis**]
El genotipo D/D del gen de la enzima conversiva de la angiotensina como factor de riesgo de reestenosis post-stent coronario.
Jul 1999

7/6/116 (Item 1 from file: 399)

A potent genetic risk factor for restenosis
?
PLEASE ENTER A COMMAND OR BE LOGGED OFF IN 5 MINUTES
? t s7/7/18, 19, 22, 28, 77

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7/7/18 (Item 18 from file: 5)

Protective role against **restenosis** from an **interleukin**-1 receptor antagonist gene **polymorphism** in patients treated with coronary stenting.
AUTHOR: Kastrati Adnan(a); Koch Werner; Berger Peter B; Mehilli Julinda; Stephenson Katherine; Neumann Franz-Josef; von Beckerath Nicolas; Boettiger Corinna; Duff Gordon W; Schoemig Albert
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JOURNAL: Journal of the American College of Cardiology 36 (7):p2168-2173 December, 2000
MEDIUM: print
ISSN: 0735-1097
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: OBJECTIVES: To test the hypothesis that **interleukin**-1 receptor antagonist (**IL**-1ra) gene **polymorphism** contributes to the risk of **restenosis** after coronary stenting. BACKGROUND: Cytokines of the **interleukin**-1 (**IL**-1) family play a central role in regulating inflammatory responses. There is strong evidence to support **IL**-1 involvement in smooth muscle cell mitogenesis and extracellular matrix metabolism. The **IL**-1ra counters the proinflammatory effects of **IL**-1. The **interleukin**-1 receptor antagonist gene (**IL**-1RN) contains several well-characterized polymorphic sites that correlate with altered **IL**-1ra levels. METHODS: In 1,850 consecutive patients, clinical and angiographic measures of **restenosis** were evaluated over one year after coronary stent placement. Repeat angiography at six months was achieved in 84% of the patients; angiographic **restenosis** was defined ltoreq50% diameter stenosis at follow-up. Genotyping for an exon 2 **polymorphism** (+2,018) of **IL**-1RN (alleles 1 and 2) was based on a polymerase chain reaction technique. RESULTS: **Allele** 2 frequency was 0.28. Carriers of **allele** 2 had a significantly lower risk for angiographic **restenosis**, odds ratio (OR) of 0.78 (95% confidence interval, 0.63 to 0.97) and target vessel revascularization, OR of 0.73 (0.58 to 0.92) compared with noncarriers. Risk reduction was especially significant in patients <60 years (n = 696), with OR of 0.63 (0.43 to 0.91) for angiographic **restenosis** and 0.55 (0.39 to 0.78) for target vessel revascularization. CONCLUSIONS: **Allele** 2 of the **IL**-1ra gene was associated with a lower incidence of **restenosis** after coronary stenting, particularly in younger patients. This finding supports a role of inflammation in the development of **restenosis** after stent placement.

12776795 BIOSIS NO.: 200000530418
Protection against **restenosis** from an **interleukin**-1 receptor antagonist gene **polymorphism** in patients treated with coronary stenting.
AUTHOR: Kastrati A; Koch W(a); Berger P B; Mehilli J(a); Stephenson K; von Beckerath N(a); Boettiger C(a); Schoemig A(a); diGiovine F; Duff G W
AUTHOR ADDRESS: (a)Deutsches Herzzentrum, TU Munich, Munich**Germany

JOURNAL: European Heart Journal 21 (Abstract Supplement):p390
August-September, 2000
MEDIUM: print
CONFERENCE/MEETING: XXII Congress of the European Society of Cardiology
Amsterdam, Netherlands August 26-30, 2000
SPONSOR: European Society of Cardiology
ISSN: 0195-668X
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

4/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12281187 BIOSIS NO.: 200000034689
Genetic risk factors for post-PTCA **restenosis**: A comprehensive analysis of multiple candidate genes.
AUTHOR: Zee Robert Y I(a); Fernandez-Ortiz Antonio; Macaya Carlos; Pintor Emilio; Fernandez-Cruz Arturo; Lindpaintner Klaus
AUTHOR ADDRESS: (a)Brigham and Women's Hosp, Harvard Med Sch, Boston, MA** USA
JOURNAL: Circulation 110 (18 SUPPL.):pI755 Nov. 2, 1999
CONFERENCE/MEETING: 72nd Scientific Sessions of the American Heart Association Atlanta, Georgia, USA November 7-10, 1999
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English

4/7/4 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

02872745 Genuine Article#: ML674 Number of References: 104
Title: VASCULAR TRANSCELLULAR SIGNALING
Author(s): MARCUS AJ; HAJJAR DP
Corporate Source: CORNELL UNIV,MED CTR,COLL MED,DEPT BIOCHEM,1300YORK AVE/NEW YORK//NY/10021; CORNELL UNIV,MED CTR,COLL MED,DEPT BIOCHEM,1300YORK AVE/NEW YORK//NY/10021; CORNELL UNIV,MED CTR,COLL MED,DEPT PATHOL/NEW YORK//NY/10021; NEW YORK VET AFFAIRS MED CTR,DIV HEMATOL ONCOL,THROMBOSIS RES LAB/NEW YORK//NY/00000
Journal: JOURNAL OF LIPID RESEARCH, 1993, V34, N12 (DEC), P2017-2031
ISSN: 0022-2275
Language: ENGLISH Document Type: REVIEW

4/7/5 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

02644125 Genuine Article#: LT743 Number of References: 40
Title: ACTIVATED ALPHA-2-MACROGLOBULIN AND TRANSFORMING GROWTH-FACTOR-BETA-1 INDUCE A SYNERGISTIC SMOOTH-MUSCLE CELL PROLIFERATIVE RESPONSE
Author(s): STOUFFER GA; LAMARRE J; GONIAS SL; OWENS GK
Corporate Source: UNIV VIRGINIA,SCH MED,DEPT MOLEC PHYSIOL & CELLULAR

BIOPHYS,BOX 449/CHARLOTTESVILLE//VA/22908; UNIV VIRGINIA,SCH MED,DEPT MOLEC PHYSIOL & CELLULAR BIOPHYS,BOX 449/CHARLOTTESVILLE//VA/22908; UNIV VIRGINIA,SCH MED,DEPT MED/CHARLOTTESVILLE//VA/22908; UNIV VIRGINIA,SCH MED,DEPT PATHOL & BIOCHEM/CHARLOTTESVILLE//VA/22908
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1993, V268, N24 (AUG 25), P 18340-18344
ISSN: 0021-9258
Language: ENGLISH Document Type: ARTICLE
Abstract: The role that soluble binding proteins might play in regulating transforming growth factor-beta1 (TGF-beta1)-induced growth of smooth muscle cells (SMC) is unknown. Alpha2-macroglobulin (alpha2M) is the major plasma binding protein for TGF-beta. Reaction of alpha2M with methylamine (alpha2M-MA) forms ''activated'' alpha2M which binds TGF-beta and specific cell surface receptors. The objectives of these studies were to determine whether native alpha2M or alpha2M-MA influences growth responses of cultured rat aortic SMC to TGF-beta1. Results demonstrated that native alpha2M was not mitogenic. Treatment with alpha2M-MA or TGF-beta1 stimulated a 3- or 3.5-fold increase in [H-3]thymidine incorporation, respectively. Cotreatment with TGF-beta1 and alpha2M-MA resulted in a 70-fold increase in [H-3]thymidine incorporation. SMC bound alpha2M-MA in a specific and saturable manner and expressed alpha2M receptor/low density lipoprotein receptor-related protein (LRP). A modified form of alpha2M-MA (alpha2M-MA-cis-dichlorodiammine platinum), which bound TGF-beta1 but did not bind alpha2M receptors, failed to enhance TGF-beta1-induced growth. In summary, results demonstrated that alpha2M-MA enhanced TGF-beta1-induced growth responses and that this effect was dependent on alpha2M-MA binding to alpha2M receptor/LRP.

4/7/6 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

10992842 EMBASE No: 2001035352
A 25-year-old with severe coronary artery disease
Ward M.R.; Herity N.A.; Lee D.P.; Yeung A.C.
Dr. A.C. Yeung, Division of Cardiovascular Medicine, Stanford University Medical Center, 300 Pasteur Drive, Stanford, CA 94305-5218 United States

AUTHOR EMAIL: alan yeung@cvmed.stanford.edu
Lancet (LANCET) (United Kingdom) 13 JAN 2001, 357/9250 (116)
CODEN: LANCA ISSN: 0140-6736
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 5

4/7/7 (Item 1 from file: 98)
DIALOG(R)File 98:General Sci Abs/Full-Text

03766792 H.W. WILSON RECORD NUMBER: BGSI98016792 (THIS IS THE FULLTEXT)
Normal and abnormal consequences of apoptosis in the human heart.
James, Thomas N
Annual Review of Physiology (Annu Rev Physiol) v. 60 ('98) p. 309-25
LANGUAGE: English
COUNTRY OF PUBLICATION: United States

WORD COUNT: 8534

ABSTRACT: Knowledge about apoptosis has become essential for understanding many aspects of cardiac structure and function. In the human heart there are major periods of morphogenesis that begin only after birth, and some of these processes recur intermittently for many years. Although the exact mechanisms by which these events are initiated or terminated remain poorly understood, it is clear that their benefits may be mirrored in destructive effects. In this review, selected examples include normal morphogenesis of the cardiac conduction system and the normal postnatal involution of the right ventricle, both of which are mediated by apoptosis. Destructive counterparts include familial heart block ending in fatal arrhythmias, similar results in the long QT syndrome, and the pathogenesis of both Uhl's anomaly and arrhythmogenic right ventricular dysplasia; in each apoptosis is an important factor. With permission, from the Annual Review of Physiology Volume 60, 1998, by Annual Reviews Inc. (http://www.annurev.org).

TEXT:

KEY WORDS: sinus node, AV node, right ventricular involution, Uhl's anomaly

INTRODUCTION

Although apoptosis remains an unfamiliar word and concept for some physicians and scientists, an understanding of apoptosis has already become essential in explaining many fundamental biological processes such as those by which a variety of cardiovascular functions and diseases emerge and evolve. The word apoptosis was introduced by Kerr, Wyllie & Currie (1) to explain a generally ignored form of cell death very different from necrosis. It is an original Greek word referring to a quiet dropping out process similar to leaves falling from a tree, an image resembling most histologic examples of apoptosis. An excellent history of earlier morphological descriptions of this type over the past century has recently been published by Majno & Joris (2), but the broad significance of apoptosis to explain many normal biological functions, such as morphogenesis and the pathogenesis of a variety of immunological and malignant diseases, only began to be recognized more widely through several perceptive interpretations by Wyllie and his colleagues (3-5).

Regrettable delay in the appreciation of major opportunities for understanding the pathogenesis of disease is nothing new. For example, the pioneering reports of Glucksmann (6), Saunders (7), and Pexeider (8), in which they emphasized that cell death was not only normal but an essential component of morphogenesis, did not receive the attention of scientists or physicians that those reports warranted. Familiar examples of useful morphogenesis include the crucial role of cell death (by apoptosis) in the fetal transformation of interdigital webs into fingers and toes, and the postnatal death of enormous numbers of neurons in the development of useful signaling pathways during normal maturation of the brain.

When I first suggested that cell death was ubiquitous and also normal in the postnatal maturation of the AV (atrioventricular) node and His bundle and that it could pose a hazard for crib death (9), given certain concurrent factors present by chance (10), the idea was harshly criticized (11-13) for a variety of stated reasons. Some said that cell death was never normal, some said that they did not see examples of such cell death in the conduction system of the heart, while still others said yes they saw exactly what I described but that it was a normal phenomenon and not one of cell death because "there was no evidence of necrosis." Apoptosis was

presumably an unfamiliar or unacceptable phenomenon for these critics. But as recently emphasized in my refutations of those criticisms, cell death is a normal component of postnatal morphogenesis of the human cardiac conduction system, and apoptosis is a major and possibly the principal mechanism by which this occurs (14).

However, the fact that apoptotic cell death may very often be useful does not mean that apoptosis is always a beneficial process. This seeming paradox--in some respects the yin and yang of cardiology--is better understood when one considers the multitude of different promoters or inhibitors of apoptosis, each of which has a progression that in some circumstances may be reversible, but at a later stage becomes irreversible, thus committing the cell inevitably to die. Unsurprisingly, the balance betwen promotion and inhibition of apoptosis often changes with time, or as a consequence of associated metabolic or toxic influences, and it differs in different organs of the mammalian body and differs to some degree between species. Whether apoptosis in the human heart is beneficial or harmful depends significantly upon when it happens and how long it lasts. It is the purpose of this review to examine both beneficial and harmful effects of apoptosis in the human heart and to briefly discuss their clinical significance.

ILLUSTRATIVE EXAMPLES OF NORMAL AND ABNORMAL CONSEQUENCES OF APOPTOSIS IN THE HUMAN HEART

POSTNATAL MORPHOGENESIS OF THE CARDIAC CONDUCTION SYSTEM

Both the sinus node and the AV conduction system (node, His bundle, and branches) can readily be recognized by the middle of the first trimester of fetal development (15, 16), but they remain essentially unchanged thereafter until one or two weeks following birth. Then the postnatal sinus node begins to be transformed (Figure 1, see color insert) from a mass of P cells, distributed about the large artery around which the sinus node is formed, into a more intricate network of slender transitional cells connecting many small groups of P cells (17, 18). The time when the sinus node achieves its adult configuration varies among individuals but is usually complete in the first few years of life. The sinus node in old age normally contains an increased volume of collagen (19), and this is sometimes misinterpreted as a pathological fibrosis. However, if the increasing fibrosis remains symmetrically distributed and the nodal myocytes similarly dispersed, this is not only normal but may even represent certain physiological advantages for impulse formation and its orderly extranodal distribution.

Also within the first week or two after birth, the fetal AV node and His bundle change morphologically very little. But an elegant experimental study by Preston, McFadden & Moe (20) some years ago demonstrated a physiological or functional immaturity of the AV conduction tissues in the very young of three different mammalian species, from which the investigators proposed a mechanism for electrical vulnerability leading to sudden unexpected death of human infants.

At about the age of two weeks, an orderly morphogenesis begins in which the irregular shape of the left side of the human AV node and His bundle [Figures 2 (see color insert) and 3] becomes transformed into a smooth border by a process of non-inflammatory resorptive degeneration (14, 18, 21). This process has now been demonstrated to include, and possibly be fully mediated by, apoptosis (14, 22). In every heart, this transforming process shapes and molds the AV node and His bundle through recurring bouts of apoptosis--weeks or months apart and not as a massive single event--and is normally complete by adolescence. Another possible morphogenetic influence in this postnatal morphogenesis is the extracellular matrix

(23-25). Actually, the sinus node is normally encased in a periarterial collagen framework, and both the AV node and His bundle are normally bounded at their margins by the collagenous central fibrous body.

The fact that the sinus node is normally encased in a collagen framework surrounding the sinus node artery led Soderstrom (26) to suggest that the sinus node resembles an enormous adventitia of its artery. Postnatal morphogenesis of the sinus node certainly includes apoptosis in a selectively distributed pattern with interweaving collagen, but it is uncertain whether that collagen acts to some degree as an inducer of apoptosis or whether there are genetically programmed or targeted nodal cells for primary apoptosis not causally related to the extracellular matrix. Just as there may be an electromechanical servomechanism (27, 28) influencing sinus impulse formation (mediated by the collagen frame of the node being attached to the sinus node artery), we can also consider the possibility that this anatomical arrangement influences how the apoptosis occurs in producing the eventual exquisite geometry of the adult sinus node's configuration. The mature interwoven pattern of a collagen matrix for the nodal myocytes (P cells and transitional cells) could be guided by physical motion determined with each phasic systolic distention (pulse) of the sinus node artery; stretch, for example, has been shown to induce apoptosis (29).

When cut in cross section (Figures 2 and 3), the fetal AV node and His bundle normally have an irregular or shaggy configuration that includes a wide dispersion of bands and clumps of their myocytes throughout the neighboring central fibrous body (18, 21). It was this disproportionately large mass of special myocytes that led Keith & Flack to refer to both structures in the fetal heart as relatively enormous (30). The orderly pruning of this surplus tissue, primarily accomplished by apoptosis (14, 22), normally leaves the AV node and His bundle as smoothly defined structures and almost certainly renders them physiologically safer for their critically important electrical function.

If fronds of normal tissue persist in the central fibrous body adjacent to the AV node, they can become the anatomical substrate for sites of either spontaneous automatic rhythm such as parasystole, or re-entrant (reciprocating) tachycardia (31). Failure in this normal molding and shaping of the AV node and His bundle may also cause unstable and hazardous electrical function there and even sudden death (32). It has recently been demonstrated that AV nodal re-entry is nearly always the specific basis of human fetal tachycardia (33). The lethal hazard of persistent fetal dispersion (in the central fibrous body) of the AV node and His bundle (32) is also supported by experimental evidence of a special postnatal electrical vulnerability of an immature AV transmission system (20).

SELECTIVE APOPTOTIC DESTRUCTION OF THE SINUS NODE AND AV NODE PLUS THE INTERNODAL AND INTERATRIAL PATHWAYS

Gradually progressive development of heart block ending with fatal arrhythmias has been described as a familial problem in South Africa (34, 35). Colleagues and I have conducted postmortem examination of the entire cardiac conduction system of a young woman who had gradually progressive development of complete heart block ending in fatal arrhythmias (22), and we found total absence of the AV node (Figure 4, see color insert) to explain the heart block, but she also had virtually no cardiac myocytes in the internodal and interatrial pathways (Figure 5) and extensive destruction of the sinus node (Figure 6, see color insert). In each of these structures there was abundant apoptosis (Figure 7, see color insert), whereas the His bundle and the myocardium of both ventricles were normal.

As part of that same investigation (22), we examined two other hearts

from five brothers with a strong family history of heart block and fatal arrhythmias, where we found similar selective apoptotic destruction of the sinus node, AV node, internodal and interatrial pathways, with sparing of the His bundle and all the ventricular myocardium. Two surviving brothers in that family were successfully treated with implanted automatic cardioverter-defibrillators, without which it seems likely that they would have died as well.

Selectivity of the apoptosis in these three hearts and the sparing of ventricular myocardium and the His bundle is perplexing, as is apparent from the following descriptions of different selectivity patterns for harmful apoptosis in other subjects with fatal arrhythmias. On the other hand, the selective destruction of the internodal and interatrial pathways, but not other atrial myocardium in each of those three hearts, lends support to the concept of some from of specialization in the internodal pathway cells (36, 37), analogous to the well-documented (38, 39) preservation of functional electrical activity among them in the presence of experimental hyperkalemia of a degree sufficient to eliminate all contractile function in other atrial myocytes, i.e. those not in the internodal and interatrial pathways.

APOPTOTIC DESTRUCTION OF MYOCYTES, VASCULAR ENDOTHELIAL AND SMOOTH MUSCLE CELLS, AND NEURAL STRUCTURES IN THE SINUS NODES OF FATAL CASES OF THE LONG QT SYNDROME

In collaboration with Russian colleagues we conducted electron microscopic examination (Figure 8) of plastic-embedded tissue obtained from sinus nodes that had been surgically excised from hearts of patients who had the long QT syndrome and intractable arrhythmias (40). Bokeriya and his surgical associates have developed a surprising protocol for successful treatment of such patients by removing the sinus node and then utilizing permanent electronic pacing (41). This seemingly draconian method is actually not so different from a widely utilized protocol of large doses of b-receptor blockers to suppress ventricular excitability but concomitantly causing a strong negative chronotropic effect, thus worsening an already dangerous sinus bradycardia, which is countered by employing permanent electronic pacing (42, 43). Actually, leaving the sinus node in place in those patients has its own hazard of ectopic or escape sinus beats causing serious electrical disturbance even in the presence of pacing (44, 45).

Sinus bradycardia in the presence of markedly delayed repolarization is conductive to a variety of potentially fatal arrhythmias (46-48). Morphological abnormalities in the sinus node in fatal cases of the long QT syndrome were the first structural pathology reported in the conduction system of such patients (46). Neither of the two original studies of the long QT syndrome (49, 50) reported any anatomical abnormalities in the hearts of their fatal cases. In another publication, we have described extensive cardioneuropathy present in the hearts of eight cases of fatal long QT syndrome, some of which was in the ventricular myocardium, but the neural abnormalities (both nerves and ganglia) were primarily in and near the sinus node (51).

Our electron microscopic studies of the sinus nodes from Russian patients demonstrated abnormal focal fibrosis in each sinus node, accompanied by narrowed small arteries and degenerated neural elements, without associated inflammation (40). In each sinus node there were many examples of typical apoptosis destroying nodal cells (Figure 8). Surprisingly, there were also numerous cells exhibiting pleomorphic micromitochondriosis, which we did not expect t

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4/7/7 (Item 1 from file: 98)
DIALOG(R)File 98:General Sci Abs/Full-Text

03766792 H.W. WILSON RECORD NUMBER: BGSI98016792 (THIS IS THE FULLTEXT)
Normal and abnormal consequences of apoptosis in the human heart.
James, Thomas N
Annual Review of Physiology (Annu Rev Physiol) v. 60 ('98) p. 309-25
LANGUAGE: English
COUNTRY OF PUBLICATION: United States
WORD COUNT: 8534

ABSTRACT: Knowledge about apoptosis has become essential for understanding many aspects of cardiac structure and function. In the human heart there are major periods of morphogenesis that begin only after birth, and some of these processes recur intermittently for many years. Although the exact mechanisms by which these events are initiated or terminated remain poorly understood, it is clear that their benefits may be mirrored in destructive effects. In this review, selected examples include normal morphogenesis of the cardiac conduction system and the normal postnatal involution of the right ventricle, both of which are mediated by apoptosis. Destructive counterparts include familial heart block ending in fatal arrhythmias, similar results in the long QT syndrome, and the pathogenesis of both Uhl's anomaly and arrhythmogenic right ventricular dysplasia; in each apoptosis is an important factor. With permission, from the Annual Review of Physiology Volume 60, 1998, by Annual Reviews Inc. (http://www.annurev.org).

TEXT:

KEY WORDS: sinus node, AV node, right ventricular involution, Uhl's anomaly

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physicians that those reports warranted. Familiar examples of useful morphogenesis include the crucial role of cell death (by apoptosis) in the fetal transformation of interdigital webs into fingers and toes, and the postnatal death of enormous numbers of neurons in the development of useful signaling pathways during normal maturation of the brain.

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ILLUSTRATIVE EXAMPLES OF NORMAL AND ABNORMAL CONSEQUENCES OF APOPTOSIS IN THE HUMAN HEART

POSTNATAL MORPHOGENESIS OF THE CARDIAC CONDUCTION SYSTEM

Both the sinus node and the AV conduction system (node, His bundle, an

03546695 H.W. WILSON RECORD NUMBER: BGSI97046695 (THIS IS THE FULLTEXT)
Cellular and molecular mechanisms of pulmonary vascular remodeling.
Stenmark, K. R
Mecham, R. P
Annual Review of Physiology (Annu Rev Physiol) v. 59 ('97) p. 89-144
LANGUAGE: English
COUNTRY OF PUBLICATION: United States
WORD COUNT: 25314

ABSTRACT: In many organs and tissues, the cellular response to injury is associated with a reiteration of specific developmental processes. Studies have shown that, in response to injury, vascular wall cells in adult organisms express genes or gene products characteristic of earlier developmental states. Other genes, expressed preferentially in adult cells in vivo, are down-regulated following injurious stimuli. Complicating matters, however, are recent observations demonstrating that the vascular wall is comprised of phenotypically heterogeneous subpopulations of endothelial cells, smooth muscle cells, and fibroblasts. It is unclear how specific subsets of cells respond to injury and thus contribute to the vascular remodeling that characterizes chronic pulmonary hypertension. This review discusses vascular development in the lung and the cellular responses occurring in pulmonary hypertension; special attention is given to heterogeneity of responses within cell populations and reiteration of developmental processes. With permission, from the Annual Review of Physiology Volume 59, 1997, by Annual Reviews Inc. (http:/www.annurev.org).

TEXT:

KEY WORDS: pulmonary hypertension, lung development, vascular development, vascular injury, smooth muscle cells, fibroblasts, endothelial cells

INTRODUCTION

In several adult tissues (e.g. liver and heart), the cellular response to injury is associated with a reiteration of specific developmental processes. This seems to be true for the vasculature as well. Studies have shown that adult smooth muscle cells (SMCs) responding to injury express genes or gene products (such as tropoelastin, fibronectin, tenascin, F31/H19, cytokeratin 8, and extra domain-A fibronectin) characteristic of earlier developmental states (76, 90, 101, 113, 134, 141). In addition, genes such as smooth muscle-specific a-actin, tropomyosin, desmin, and myosin, which are expressed preferentially in adult cells in vivo, are down-regulated when adult cells are stimulated to migrate and divide following injurious stimuli (116, 117). Similar though less extensive reports exist regarding re-expression of developmentally regulated genes by endothelial cells and fibroblasts in response to injury. Complicating matters, however, are recent observations demonstrating that the vascular media is comprised of phenotypically heterogeneous subpopulations of SMCs. It is unclear if all adult medial SMC subpopulations are capable of responding to injury with changes in gene expression and replicative potential, or if the post-injury response is limited to specific subsets of cells within the vessel wall (222). The goal of this review is thus twofold: (a) to discuss vascular development in the lung, especially the mechanisms that control growth and differentiation (in an effort to lay the groundwork for understanding the cellular responses and the mechanisms that control them in the setting of pulmonary vascular injury) and (b) to discuss the cellular changes that occur in various forms of pulmonary hypertension, with special attention to heterogeneity of responses within cell populations and reiteration of developmental processes (Figure 1).

DEVELOPMENT OF THE PULMONARY VASCULATURE

The lung is a highly complex organ comprised of more than 40 different cell types (38) that are involved in both respiratory and nonrespiratory functions. Despite its eventual complexity of structure and function, the lung has ostensibly simple beginnings. The lung epithelium originates as paired outpocketings from the floor of the pharyngeal endoderm that expand

into mesenchyme derived from splanchnic mesoderm. The epithelial rudiments subsequently undergo a series of repetitive branchings to give rise to the pulmonary tree. Much effort has been directed at defining the mechanisms that regulate normal epithelial pattern formation (termed branching morphogenesis), but much less is known about how the pulmonary vasculature is formed. The development and maintenance of normal vascular structure clearly plays a critical role in lung function, yet several major questions about the regulation of this process in the normal and diseased lung remain unanswered.

ENDOTHELIAL CELL REPLICATION AND VASCULOGENESIS IN THE DEVELOPING LUNG
In the embryonic lung, endothelial precursor cells (angioblasts) initially form a primary vascular plexus within the tissue, which eventually links up to the main circulation, of sixth branchial arch origin, coming from the heart. Studies using intracoelomic chimeric recombinations between quail and chick embryonic lungs have demonstrated that the endoderm seems to control lung vasculogenesis by inducing the emergence of endothelial cells in its associated mesoderm (184). Furthermore, tissue mixing experiments also suggest that vessel formation in the lung, like branching morphogenesis, may depend on the interaction between epithelium and mesenchyme (129, 143, 212). Thus a true reciprocity would exist in which epithelial proliferation and differentiation are induced by an as-yet-unidentified mesenchymal cell population. The induced epithelium (or a subpopulation of cells thereof) in turn produces factors that stimulate endothelial cell proliferation and organization. The endothelium itself may then produce factors to recruit other mesenchymal cells (e.g. SMC precursors) necessary for the completion of vessel structure.

Specific growth factors found in the developing lung, including acidic fibroblast growth factor (aFGF), basic fibroblast growth factor (bFGF), and platelet-derived endothelial cell growth factor (PD-ECGF), regulate endothelial cell proliferation in vivo and in vitro (81, 84, 99, 216). Another candidate molecule likely to play a major role in lung vascular development is vascular endothelial growth factor (VEGF), a member of the PDGF family that is identical to vascular permeability factor (157) and vasculotropin (190). It is abundant in highly vascularized tissues such as kidney, placenta, and lung. Unlike the FGFs, VEGF appears to be an endothelial-specific mitogen and is involved in both
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4/7/9 (Item 1 from file: 144)
DIALOG(R)File 144:Pascal

13863741 PASCAL No.: 99-0041435
Influence des alleles de l'apolipoproteine E sur la restenose apres angioplastie coronaire chez la femme
(Influence of apolipoprotein E alleles on post-angioplasty **restenosis** in woman)
FLORK L; JOUANEL P; LUSSON J R; LEAUTE S; DAUPHIN C; MOTREFF P; JUSTIN E P; LAMAISON D; BOIRE J Y; CASSAGNES J
Service de cardiologie, CHU G-Montpied, 63009 Clermond-Ferrand, France; Laboratoire de biochimie, CHU Hotel-Dieu, 63009 Clermond-Ferrand, France;

ERIM, Faculte de medecine, BP 38, 63001 Clermond-Ferrand, France
Journal: Archives des maladies du coeur et des vaisseaux, 1998, 91 (12) 1475-1479
ISSN: 0003-9683 CODEN: AMCVAN Availability: INIST-887; 354000073231900050
No. of Refs.: 23 ref.
Document Type: P (Serial) ; A (Analytic)
Country of Publication: France
Language: French

Meme si l'emploi d'endoprothese en diminue l'incidence, le phenomene de restenose coronaire apres dilatation percutanee reste entier. L'influence des lipoproteines dans le developpement de l'atherosclerose n'est plus a demontrer, mais leur role dans la restenose est plus discutable. Des resultats contradictoires ont ete publies a propos de la responsabilite du genotype de l'Apo E. Dans un premier travail, nous avons mis en evidence la relation LP (a) et coronaropathie plus etroite chez la femme que chez l'homme, Un sous-groupe de patientes qui ont eu une angioplastie dont le profil lipidique etait bien etabli a ete analyse en se focalisant sur les alleles de l'Apo E. Methodes : 59 patientes ont eu une angioplastie parmi lesquelles 35 monotronculaires, 20 bitronculaires et 4 tritronculaires. Une coronarographie de controle a ete pratiquee chez 40 patientes. Un interrogatoire telephonique a ete realise entre 12 et 22 mois apres la dilatation a l'ensemble de la population. Les apolipoproteines A1, B. LP (a) et LP A1 sont dosees par techniques immunologiques, turbidimetriques ou electro-immunologiques. Le genotypage de l'Apo E est realise a l'aide du kit Inno-Lipa. Resultats : la repartition des patientes est la suivante : 18 restenoses angiographiques (groupe A), 20 coronarographies sans restenose (groupe B), 41 sans restenose angiographique (20) ou clinique (21) (groupe C). Dans le groupe A, la LP (a) depasse largement le seuil de 0,30 g/L. L'**allele** e4 est associe a un cholesterol total et sa fraction LDL les plus eleves. **Il** n'y a pas de difference significative entre le genotype Apo E des differents groupes ni en fonction de la severite des lesions. En conclusion : si l'**allele** e4 est plus frequemment associe a un taux eleve de LDL cholesterol et LP (a), nous n'avons pas prouve son role dans le processus de restenose. Un nombre de malades plus Important est necessaire et des travaux complementaires souhaitables pour comprendre les mecanismes inflammatoires et/ou immunologiques par lesquels l'Apo E pourrait Intervenir dans la restenose.

? t s4/7/10-15

>>>Format 7 is not valid in file 143

4/7/10 (Item 2 from file: 144)
DIALOG(R)File 144:Pascal

13475066 PASCAL No.: 98-0172121
EPIDEMIOLOGIE DE LA MALADIE CORONAIRE : RECHERCHE DES DETERMINANTS GENETIQUES DE LA VASOMOTRICITE CORONAIRE ET DE LA RESTENOSE
(CORONARY DISEASE EPIDEMIOLOGY : CORONARY AND **RESTENOSIS** GENETIC RISK FACTORS DETERMINATION)
AMANT Carole; AMOUYEL Philippe, dir
Universite de Lille 1, Villeneuve-d'Ascq, Francee
Univ.: Universite de Lille 1. Villeneuve-d'Ascq. FRA Degree: Th. doct.
1997-12; 1997 183 p.
Availability: INIST-T 116002; T97LIL10134 0000; RBCCN-590092102;

T97LIL10134 0000
No. of Refs.: 214 ref.
Document Type: T (Thesis) ; M (Monographic)
Country of Publication: France
Language: French Summary Language: French; English
L'insuffisance coronaire et l'infarctus du myocarde sont les premieres causes de mortalite et de morbidite dans les pays industrialises. Comme dans la plupart des maladies multifactorielles, de nombreux facteurs de risque predisposent au developpement et a la survenue de la maladie coronaire. **Il** s'agit des facteurs de risque environnementaux et des facteurs de susceptibilite genetique. Dans le travail presente ici, nous nous sommes focalises sur des genes candidats issus du systeme renine-angiotensine. Les etudes developpees ont consiste a mesurer le degre d'implication de ce systeme dans les phenomenes de restenose et de vasoconstriction accrue. Nos resultats suggerent que l'**allele** D du gene de l'Enzyme de Conversion de l'Angiotensine I (ECA) constitue un facteur de risque majeur d'occlusion apres angioplastie conventionnelle et de restenose apres angioplastie avec pose d'endoprothese coronaire, le stent. Nous avons aussi mis en evidence que l'**allele** C du gene du recepteur de l'angiotensine II de type 1 (AT1) est lie a une vasoconstriction coronaire augmentee. Cliniquement, ces resultats sont interessants car ils permettent d'identifier les patients a haut risque de restenose et/ou de vasoconstriction augmentee dans le but de prevenir les accidents coronaires graves qui pourraient survenir, tels qu'un infarctus post-angioplastie ou un vasospasme. Cependant d'autres etudes sont necessaires pour verifier nos resultats tant au niveau epidemiologique qu'aux niveaux clinique et physiologique.

4/7/11 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11567561 21405569 PMID: 11514491
Interleukin 1 receptor antagonist gene **polymorphism** and **restenosis** after coronary angioplasty.
Francis SE; Camp NJ; Burton AJ; Dewberry RM; Gunn J; Stephens-Lloyd A; Cumberland DC; Gershlick A; Crossman DC
Cardiovascular Medicine Group, Division of Clinical Sciences, Clinical Sciences Centre, University of Sheffield, Northern General Hospital, Sheffield S5 7AU, UK.
Heart (England) Sep 2001, 86 (3) p336-40, ISSN 1468-201X Journal Code: CEN
Languages: ENGLISH
Document type: Journal Article
Record type: In Process
BACKGROUND: Percutaneous transluminal coronary angioplasty (PTCA) is limited by the recurrence of luminal stenosis, which occurs in up to 50% of procedures. It has been shown that patient specific factors, perhaps genes, contribute to this process. OBJECTIVE: To determine whether completion of healing after PTCA is part of an acute self limiting inflammatory process and whether **polymorphism** at important inflammatory gene loci might determine susceptibility to **restenosis** after PTCA. DESIGN: DNA samples were collected from 171 patients attending for elective PTCA in Sheffield (S) and Leicester (L), who were scheduled to undergo follow up angiography (at four months (L) or six months (S)) as part of other **restenosis** studies. At follow up angiography, the patients were

separated into restenosers (> 50% luminal narrowing) and non-restenosers (< 50% luminal narrowing). Four DNA polymorphisms within **interleukin** 1 (**IL**-1) related loci (**IL**-1A (-889), **IL**-1B (-511), **IL** -1B (+3954), and **IL** -1RN intron 2 VNTR (variable number tandem repeat)) were genotyped using methods based on polymerase chain reaction. Significance was assessed by chi(2) analysis of the relevant contingency table, and the magnitude of effect was estimated by calculating odds ratios. The Mantel-Haenszel (MH) test was applied to summarise data across the two populations. RESULTS: **Allele** 2 at **IL**-1RN (**IL** -1RN*2) was significantly over represented in the non-restenoser group (L+S, 34% v 23% in restenosers). Furthermore, **IL**-1RN*2 homozygosity was increased in the non-restenoser population compared with the restenosers (MH test: p = 0.0196 (L+S); p = 0.031 (L+S, single vessel disease only), and the effect seemed to be restricted to the single vessel disease subpopulation. For other **polymorphism** within **IL**-1 related loci no significant associations were found with either **restenosis** or non-**restenosis**. CONCLUSIONS: **IL**-1RN*2 may be associated with protection from **restenosis** after PTCA for individuals with single vessel disease. As this **polymorphism** has functional significance, this finding suggests that alteration in an individual's inflammatory predisposition may modulate the blood vessel response to injury.

Record Date Created: 20010821

4/7/12 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

135032750 CA: 135(3)32750s PATENT
Interleukin-1 homologue, MAT IL-1H4
INVENTOR(AUTHOR): Kumar, Sanjay; McDonnell, Peter C.; Young, Peter R.
LOCATION: USA
ASSIGNEE: Smithkline Beecham Corporation
PATENT: PCT International ; WO 0140247 A1 DATE: 20010607
APPLICATION: WO 2000US32521 (20001130) *US 452140 (19991201)
PAGES: 30 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C07H-021/02A; C12N-015/00B; C12N-005/00B; C07K-001/00B; C07K-016/00B
DESIGNATED COUNTRIES: JP DESIGNATED REGIONAL: AT; BE; CH; CY; DE; DK; ES ; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; TR
SECTION:
CA215005 Immunochemistry
CA201XXX Pharmacology
CA203XXX Biochemical Genetics
CA209XXX Biochemical Methods
CA214XXX Mammalian Pathological Biochemistry
IDENTIFIERS: MAT interleukin 1H4 agonist antagonist screening, antibody inflammation mature interleukin 1H4 inhibitor
DESCRIPTORS:
Respiratory distress syndrome...
acute; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others
Diagnosis...
agents; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others
Immunoassay...

enzyme-linked immunosorbent assay; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Heart,disease...
failure; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Transplant and Transplantation...
graft-vs.-host reaction; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Intestine,disease...
inflammatory; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Brain,disease...
injury; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

AIDS(disease)... Allergy... Alzheimer's disease... Antibodies... Arthritis ... Asthma... Atherosclerosis... Autoimmune disease... Bone,disease... cDNA sequences... Drug screening... Fusion proteins(chimeric proteins)... Infection... Inflammation... Ischemia... Labels... Lymphoproliferative disorders... Molecular cloning... mRNA... Mutation... Neoplasm... Nucleic acid hybridization... Oligonucleotides... Osteoporosis... Protein sequences ... Psoriasis... Septicemia... Stress,animal... Susceptibility(genetic)...

Transplant rejection...
interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Probes(nucleic acid)...
labeled; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Artery,disease...
restenosis; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Brain,disease...
stroke; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Interleukins...
1H4; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

CAS REGISTRY NUMBERS:

265295-30-5 343914-85-2 amino acid sequence; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

175941-97-6 209511-54-6 224113-70-6 interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

303170-58-3 nucleotide sequence; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

343916-05-2 343916-06-3 unclaimed nucleotide sequence; interleukin-1 homolog, MAT IL-1H4

```
      S2      584  S1 AND (POLYMORPHISM OR ALLELE OR MUTATION)
? s s2 and (interleukin or IL)

             584  S2
          725902  INTERLEUKIN
          878382  IL
      S3       24  S2 AND (INTERLEUKIN OR IL)
? rd s3

...completed examining records
      S4       15  RD S3 (unique items)
? t s4/7/1-15

>>>Format 7 is not valid in file 143

 4/7/1      (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

12815465   BIOSIS NO.: 200100022614
Protective role against restenosis from an interleukin-1
  receptor antagonist gene polymorphism in patients treated with
  coronary stenting.
AUTHOR: Kastrati Adnan(a); Koch Werner; Berger Peter B; Mehilli Julinda;
  Stephenson Katherine; Neumann Franz-Josef; von Beckerath Nicolas;
  Boettiger Corinna; Duff Gordon W; Schoemig Albert
AUTHOR ADDRESS: (a)Deutsches Herzzentrum, Muenchen, Lazarettstrasse 36,
  80636, Muenchen: kastrati@dhm.mhn.de**Germany
JOURNAL: Journal of the American College of Cardiology  36 (7):p2168-2173
December, 2000
MEDIUM: print
ISSN: 0735-1097
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: OBJECTIVES: To test the hypothesis that interleukin-1
  receptor antagonist (IL-1ra) gene polymorphism contributes to
  the risk of restenosis after coronary stenting. BACKGROUND:
  Cytokines of the interleukin-1 (IL-1) family play a central
  role in regulating inflammatory responses. There is strong evidence to
  support IL-1 involvement in smooth muscle cell mitogenesis and
  extracellular matrix metabolism. The IL-1ra counters the
  proinflammatory effects of IL-1. The interleukin-1 receptor
  antagonist gene (IL-1RN) contains several well-characterized
  polymorphic sites that correlate with altered IL-1ra levels.
  METHODS: In 1,850 consecutive patients, clinical and angiographic
  measures of restenosis were evaluated over one year after coronary
  stent placement. Repeat angiography at six months was achieved in 84% of
  the patients; angiographic restenosis was defined ltoreq50%
  diameter stenosis at follow-up. Genotyping for an exon 2
  polymorphism (+2,018) of IL-1RN (alleles 1 and 2) was based
  on a polymerase chain reaction technique. RESULTS: Allele 2
  frequency was 0.28. Carriers of allele 2 had a significantly lower
  risk for angiographic restenosis, odds ratio (OR) of 0.78 (95%
  confidence interval, 0.63 to 0.97) and target vessel revascularization,
  OR of 0.73 (0.58 to 0.92) compared with noncarriers. Risk reduction was
```

especially significant in patients <60 years (n = 696), with OR of 0.63 (0.43 to 0.91) for angiographic **restenosis** and 0.55 (0.39 to 0.78) for t rget vessel revascularization. CONCLUSIONS: **Allele** 2 of the **IL**-1 gene was associated with a lower incidence of **reste sis** after coronary stenting, particularly in younger patie. s. This finding supports a role of inflammation in the development of **re: enosis** after stent placement.

4/7/2 (Item 2 from file: 5)
DIALOG(F File 5:Biosis Previews(R)
(c) 200] BIOSIS. All rts. reserv.

12776795 BIOSIS NO.: 200000530418
Protecti n against **restenosis** from an **interleukin**-1 receptor antago ist gene **polymorphism** in patients treated with coronary stenti g.
AUTHOR: astrati A; Koch W(a); Berger P B; Mehilli J(a); Stephenson K; von Becke th N(a); Boettiger C(a); Schoemig A(a); diGiovine F; Duff G W
AUTHOR A DRESS: (a)Deutsches Herzzentrum, TU Munich, Munich**Germany
JOURNAL: European Heart Journal 21 (Abstract Supplement):p390 August-S ptember, 2000
MEDIUM: rint
CONFEREN E/MEETING: XXII Congress of the European Society of Cardiology Amsterda , Netherlands August 26-30, 2000
SPONSOR: uropean Society of Cardiology
ISSN: 019 -668X
RECORD TY E: Citation
LANGUAGE: English
SUMMARY L NGUAGE: English

4/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

12281187 BIOSIS NO.: 200000034689
Genetic risk factors for post-PTCA **restenosis**: A comprehensive analysis of multiple candidate genes.
AUTHOR: Zee Robert Y I(a); Fernandez-Ortiz Antonio; Macaya Carlos; Pintor Emilio; Fernandez-Cruz Arturo; Lindpaintner Klaus
AUTHOR ADDRESS: (a)Brigham and Women's Hosp, Harvard Med Sch, Boston, MA** USA
JOURNAL: Circulation 110 (18 SUPPL.):pI755 Nov. 2, 1999
CONFERENCE/MEETING: 72nd Scientific Sessions of the American Heart Association Atlanta, Georgia, USA November 7-10, 1999
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English

4/7/4 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2001 Inst for Sci Info. All rts. reserv.

02872745 Genuine Article#: ML674 Number of References: 104
Title: VASCULAR TRANSCELLULAR SIGNALING
Author(s): MARCUS AJ; HAJJAR DP

Corporate Source: CORNELL UNIV,MED CTR,COLL MED,DEPT BIOCHEM,1300YORK AVE/NEW YORK//NY/10021; CORNELL UNIV,MED CTR,COLL MED,DEPT BIOCHEM,1300YORK AVE/NEW YORK//NY/10021; CORNELL UNIV,MED CTR,COLL MED,DEPT PATHOL/NEW YORK//NY/10021; NEW YORK VET AFFAIRS MED CTR,DIV HEMATOL ONCOL,THROMBOSIS RES LAB/NEW YORK//NY/00000
Journal: JOURNAL OF LIPID RESEARCH, 1993, V34, N12 (DEC), P2017-2031
ISSN: 0022-2275
Language: ENGLISH Document Type: REVIEW

4/7/5 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2001 Inst for Sci Info. All rts. reserv.

02644125 Genuine Article#: LT743 Number of References: 40
Title: ACTIVATED ALPHA-2-MACROGLOBULIN AND TRANSFORMING GROWTH-FACTOR-BETA-1 INDUCE A SYNERGISTIC SMOOTH-MUSCLE CELL PROLIFERATIVE RESPONSE
Author(s): STOUFFER GA; LAMARRE J; GONIAS SL; OWENS GK
Corporate Source: UNIV VIRGINIA,SCH MED,DEPT MOLEC PHYSIOL & CELLULAR BIOPHYS,BOX 449/CHARLOTTESVILLE//VA/22908; UNIV VIRGINIA,SCH MED,DEPT MOLEC PHYSIOL & CELLULAR BIOPHYS,BOX 449/CHARLOTTESVILLE//VA/22908; UNIV VIRGINIA,SCH MED,DEPT MED/CHARLOTTESVILLE//VA/22908; UNIV VIRGINIA,SCH MED,DEPT PATHOL & BIOCHEM/CHARLOTTESVILLE//VA/22908
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1993, V268, N24 (AUG 25), P 18340-18
ISSN: 0021-9
Language: EN Document Type: ARTICLE
Abstract: The possible that soluble binding proteins might play in regulating transforming growth factor-beta1 (TGF-beta1)-induced growth of smooth muscle cells (SMC) is unknown. Alpha2-macroglobulin (alpha2M) is the major plasma binding protein for TGF-beta. Reaction of alpha2M with methylamine (alpha2M-MA) forms ''activated'' alpha2M which binds TGF-beta and specific cell surface receptors. The objectives of these studies were to determine whether native alpha2M or alpha2M-MA influences growth responses of cultured rat aortic SMC to TGF-beta1. Results demonstrated that native alpha2M was not mitogenic. Treatment with alpha2M-MA or TGF-beta1 stimulated a 3- or 3.5-fold increase in [H-3]thymidine incorporation, respectively. Cotreatment with TGF-beta1 and alpha2M-MA resulted in a 70-fold increase in [H-3]thymidine incorporation. SMC bound alpha2M-MA in a specific and saturable manner and expressed alpha2M receptor/low density lipoprotein receptor-related protein (LRP). A modified form of alpha2M-MA (alpha2M-MA-cis-dichlorodiammine platinum), which bound TGF-beta1 but did not bind alpha2M receptors, failed to enhance TGF-beta1-induced growth. In summary, results demonstrated that alpha2M-MA enhanced TGF-beta1-induced growth responses and that this effect was dependent on alpha2M-MA binding to alpha2M receptor/LRP.

4/7/6 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
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10992842 EMBASE No: 2001035352
A 25-year-old with severe coronary artery disease
Ward M.R.; Herity N.A.; Lee D.P.; Yeung A.C.
Dr. A.C. Yeung, Division of Cardiovascular Medicine, Stanford University

Medical Center, 300 Pasteur Drive, Stanford, CA 94305-5218 United States

AUTHOR EMAIL: alan yeung@cvmed.stanford.edu
Lancet (LANCET) (United Kingdom) 13 JAN 2001, 357/9250 (116)
CODEN: LANCA ISSN: 0140-6736
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 5

03766792 H.W. WILSON RECORD NUMBER: BGSI98016792 (THIS IS THE FULLTEXT)
Normal and abnormal consequences of apoptosis in the human heart.
James, Thomas N
Annual Review of Physiology (Annu Rev Physiol) v. 60 ('98) p. 309-25
LANGUAGE: English
COUNTRY OF PUBLICATION: United States
WORD COUNT: 8534

ABSTRACT: Knowledge about apoptosis has become essential for understanding many aspects of cardiac structure and function. In the human heart there are major periods of morphogenesis that begin only after birth, and some of these processes recur intermittently for many years. Although the exact mechanisms by which these events are initiated or terminated remain poorly understood, it is clear that their benefits may be mirrored in destructive effects. In this review, selected examples include normal morphogenesis of the cardiac conduction system and the normal postnatal involution of the right ventricle, both of which are mediated by apoptosis. Destructive counterparts include familial heart block ending in fatal arrhythmias, similar results in the long QT syndrome, and the pathogenesis of both Uhl's anomaly and arrhythmogenic right ventricular dysplasia; in each apoptosis is an important factor. With permission, from the Annual Review of Physiology Volume 60, 1998, by Annual Reviews Inc. (http://www.annurev.org).

TEXT:

KEY WORDS: sinus node, AV node, right ventricular involution, Uhl's anomaly

INTRODUCTION

Although apoptosis remains an unfamiliar word and concept for some physicians and scientists, an understanding of apoptosis has already become essential in explaining many fundamental biological processes such as those by which a variety of cardiovascular functions and diseases emerge and evolve. The word apoptosis was introduced by Kerr, Wyllie & Currie (1) to explain a generally ignored form of cell death very different from necrosis. It is an original Greek word referring to a quiet dropping out process similar to leaves falling from a tree, an image resembling most histologic examples of apoptosis. An excellent history of earlier morphological descriptions of this type over the past century has recently been published by Majno & Joris (2), but the broad significance of apoptosis to explain many normal biological functions, such as morphogenesis and the pathogenesis of a variety of immunological and malignant diseases, only began to be recognized more widely through several perceptive interpretations by Wyllie and his colleagues (3-5).

Regrettable [illegible] the appreciation of major opportunities for understanding th[illegible]ogenesis of disease is nothing new. For example, the pioneering repo[illegible]lucksmann (6), Saunders (7), and Pexeider (8), in which they empha[illegible] cell death was not only normal but an essential component of mor[illegible], did not receive the attention of scientists or physicians that [illegible]eports warranted. Familiar examples of useful morphogenesis in [illegible]he crucial role of cell death (by apoptosis) in the fetal transforma[illegible] interdigital webs into fingers and toes, and the postnatal death [illegible]mous numbers of neurons in the development of useful signaling pathw[illegible] normal maturation of the brain.

When I fir[illegible]ested that cell death was ubiquitous and also normal in the postnatal [illegible]tion of the AV (atrioventricular) node and His bundle and that [illegible]d pose a hazard for crib death (9), given certain concurrent factors [illegible]sent by chance (10), the idea was harshly criticized (11-13) for a varie[illegible] stated reasons. Some said that cell death was never normal, some sa[illegible]t they did not see examples of such cell death in the conduction syst[illegible] the heart, while still others said yes they saw exactly what I describe[illegible] that it was a normal phenomenon and not one of cell death because "the[illegible] no evidence of necrosis." Apoptosis was presumably an unfamilia[illegible]acceptable phenomenon for these critics. But as recently emphasized[illegible] refutations of those criticisms, cell death is a normal component of [illegible]al morphogenesis of the human cardiac conduction system, and[illegible]osis is a major and possibly the principal mechanism by which this[illegible]s (14).

However, the fact[illegible]poptotic cell death may very often be useful does not mean that apop[illegible] is always a beneficial process. This seeming paradox--in some respec[illegible]e yin and yang of cardiology--is better understood when one co[illegible]s the multitude of different promoters or inhibitors of apoptos[illegible]n of which has a progression that in some circumstances may be [illegible]e, but at a later stage becomes irreversible, thus committing the cel[illegible]itably to die. Unsurprisingly, the balance betwen promotion and in[illegible]on of apoptosis often changes with time, or as a consequence of associ[illegible] metabolic or toxic influences, and it differs in different organs of [illegible]mammalian body and differs to some degree between species. Whethe[illegible]optosis in the human heart is beneficial or harmful depends signifi[illegible]ly upon when it happens and how long it lasts. It is the purpose of th[illegible]eview to examine both beneficial and harmful effects of apoptosis in[illegible] human heart and to briefly discuss their clinical significance.

ILLUSTRATIVE EXAMPLES OF NORMAL AND ABNORMAL CONSEQUENCES OF APOPTOSIS IN THE HUMAN HEART

POSTNATAL MORPHOGENESIS OF THE CARDIAC CONDUCTION SYSTEM

Both the sinus node and the AV conduction system (node, His bundle, and branches) can readily be recognized by the middle of the first trimester of fetal development (15, 16), but they remain essentially unchanged thereafter until one or [illegible] weeks following birth. Then the postnatal sinus node begins to be transformed (Figure 1, see color insert) from a mass of P cells, distributed about a large artery around which the sinus node is formed, into a more intricate network of slender transitional cells connecting many small groups of P cells (17, 18). The time when the sinus node achieves its adult configuration varies among individuals but is usually complete in the first few years of life. The sinus node in old age normally contains an increased volume of collagen (19), and this is sometimes misinterpreted as a pathological fibrosis. However, if the increasing fibrosis remains symmetrically distributed and the nodal myocytes similarly disposed, this is not only normal but may even

represent certain physi ical advantages for impulse formation and its orderly extranodal dist tion.

Also within the fi week or two after birth, the fetal AV node and His bundle change morpl gically very little. But an elegant experimental study by Preston, McFa & Moe (20) some years ago demonstrated a physiological or funct: l immaturity of the AV conduction tissues in the very young of three di: ent mammalian species, from which the investigators proposed echanism for electrical vulnerability leading to sudden unexpected death human infants.

At about the age c weeks, an orderly morphogenesis begins in which the irregular sha f the left side of the human AV node and His bundle [Figures 2 (see r insert) and 3] becomes transformed into a smooth border by a proc of non-inflammatory resorptive degeneration (14, 18, 21). This process h ow been demonstrated to include, and possibly be fully mediated by, apop s (14, 22). In every heart, this transforming process shapes and mold e AV node and His bundle through recurring bouts of apoptosis--weeks or is apart and not as a massive single event--and is normally complete by lescence. Another possible morphogenetic influence in this postn morphogenesis is the extracellular matrix (23-25). Actually, the s node is normally encased in a periarterial collagen framework, and the AV node and His bundle are normally bounded at their margin the collagenous central fibrous body.

The fact that the s node is normally encased in a collagen framework surrounding t inus node artery led Soderstrom (26) to suggest that the sinus node res es an enormous adventitia of its artery. Postnatal morphogenesis the sinus node certainly includes apoptosis in a selectively distribute ttern with interweaving collagen, but it is uncertain whether that collagen acts to some degree as an inducer of apoptosis or whether there are genetically programmed or targeted nodal cells for primary apoptosis not causally related to the extracellular matrix. Just as there may be an electromechanical servomechanism (27, 28) influencing sinus impulse formation (mediated by the collagen frame of the node being attached to h sinus node artery), we can also consider the possibility that this a ical arrangement influences how the apoptosis occurs in producing the tual exquisite geometry of the adult sinus node's configuration. T ture interwoven pattern of a collagen matrix for the nodal myocyte: lls and transitional cells) could be guided by physical motion determ ith each phasic systolic distention (pulse) of the sinus node artery; ch, for example, has been shown to induce apoptosis (29).

When cut in cros ion (Figures 2 and 3), the fetal AV node and His bundle normally have an egular or shaggy configuration that includes a wide dispersion of banc d clumps of their myocytes throughout the neighboring central fib body (18, 21). It was this disproportionately large mass of special m at led Keith & Flack to refer to both structures in the fet relatively enormous (30). The orderly pruning of this surplu rimarily accomplished by apoptosis (14, 22), normally leaves th e and His bundle as smoothly defined structures and almost cer renders them physiologically safer for their critically importan trical function.

If fronds of normal persist in the central fibrous body adjacent to the AV node, t an become the anatomical substrate for sites of either spontaneous aut rhythm such as parasystole, or re-entrant (reciprocating) tachycar 1). Failure in this normal molding and shaping of the AV node a bundle may also cause unstable and hazardous electrical function ther n sudden death (32). It has recently been demonstrated that AV nod try is nearly always the specific basis of

human fetal tachycardia (33). The lethal hazard of persistent fetal dispersion (in the central fibrous body) of the AV node and His bundle (32) is also supported by experimental evidence of a special postnatal electrical vulnerability of an immature AV transmission system (20).

SELECTIVE APOPTOTIC DESTRUCTION OF THE SINUS NODE AND AV NODE PLUS THE INTERNODAL AND INTERATRIAL PATHWAYS

Gradually progressive development of heart block ending with fatal arrhythmias has been described as a familial problem in South Africa (34, 35). Colleagues and I have conducted postmortem examination of the entire ...c conduction system of a young woman who had gradually progressive ...lopment of complete heart block ending in fatal arrhythmias (22), and we ...und total absence of the AV node (Figure 4, see color insert) to explain the heart block, but she also had virtually no cardiac myocytes in the internodal and interatrial pathways (Figure 5) and extensive destruction of the sinus node (Figure 6, see color insert). In each of these structures there was abundant apoptosis (Figure 7, see color insert), whereas the His bundle and the myocardium of both ventricles were normal.

...that same investigation (22), we examined two other hearts from ...others with a strong family history of heart block and fatal arrhythm... ...e we found similar selective apoptotic destruction of the sinus node, ...node, internodal and interatrial pathways, with sparing of the His bundl... ...the ventricular myocardium. Two surviving brothers in that family were successfully treated with implanted automatic cardioverter-defibrillators, without which it seems likely that they would have died as well.

Selectivity of the apoptosis ... three hearts and the sparing of ventricular myocardium and the Hi... ...xing, as is apparent from the following descriptions of different selectivity patterns for harmful apoptosis in other subjects with fatal arrhythmias. On the other hand, the selective destruction of the internodal and interatrial pathways, but not other atrial myocardium in each of those three hearts, lends support to the concept of some from of specialization in the internodal pathway cells (36, 37), analogous to the well-documented (38, 39) preservation of functional electrical activity among them in the presence of experimental hyperkalemia of a degree sufficient to eliminate all contractile function in other atrial myocytes, i.e. those not in the internodal and interatrial pathways.

APOPTOTIC DESTRUCTION OF MYOCYTES, VASCULAR ENDOTHELIAL AND SMOOTH MUSCLE CELLS, AND NEURAL STRUCTURES IN THE SINUS NODES OF FATAL CASES OF THE LONG QT SYNDROME

In collaboration with Russian colleagues we conducted electron microscopic examination (Figure 8) of plastic-embedded tissue obtained from sinus nodes that had been surgically excised from hearts of patients who had the long QT syndrome and intractable arrhythmias (40). Bokeriya and his surgical associates have developed a surprising protocol for successful treatment of such patients by removing the sinus node and then utilizing permanent electronic pacing (41). This seemingly draconian method is actually not so different from a widely utilized protocol of large doses of b-receptor blockers to suppress ventricular excitability but concomitantly causing a strong negative chronotropic effect, thus worsening an already dangerous sinus bradycardia, which is countered by employing permanent electronic pacing (42, 43). Actually, leaving the sinus node in place in those patients has its own hazard of ectopic or escape sinus beats causing serious electrical disturbance even in the presence of pacing (44, 45).

Sinus bradycardia in the presence of markedly delayed repolarization is conductive to a variety of potentially fatal arrhythmias (46-48). Morphological abnormalities in the sinus node in fatal cases of the long QT syndrome were the first structural pathology reported in the conduction system of such patients (46). Neither of the two original studies of the long QT syndrome (49, 50) reported any anatomical abnormalities in the hearts of their fatal cases. In another publication, we have described extensive cardioneuropathy present in the hearts of eight cases of fatal long QT syndrome, some of which was in the ventricular myocardium, but the neural abnormalities (both nerves and ganglia) were primarily in and near the sinus node (51).

Our electron microscopic studies of the sinus nodes from Russian patients demonstrated abnormal focal fibrosis in each sinus node, accompanied by narrowed small arteries and degenerated neural elements, without associated inflammation (40). In each sinus node there were many examples of typical apoptosis destroying nodal cells (Figure 8). Surprisingly, there were also numerous cells exhibiting pleomorphic micromitochondriosis, which we did not expect t
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4/7/7 (Item 1 from file: 98)
DIALOG(R)File 98:General Sci Abs/Full-Text

03766792 H.W. WILSON RECORD NUMBER: BGSI98016792 (THIS IS THE FULLTEXT)
Normal and abnormal consequences of apoptosis in the human heart.
James, Thomas N
Annual Review of Physiology (Annu Rev Physiol) v. 60 ('98) p. 309-25
LANGUAGE: English
COUNTRY OF PUBLICATION: United States
WORD COUNT: 8534

ABSTRACT: Knowledge about apoptosis has become essential for understanding many aspects of cardiac structure and function. In the human heart there are major periods of morphogenesis that begin only after birth, and some of these processes recur intermittently for many years. Although the exact mechanisms by which these events are initiated c[illegible]d remain poorly understood, it is clear that their benefits r[illegible]in destructive effects. In this review, selected examples [illegible]phogenesis of the cardiac conduction system and the no[illegible]tion of the right ventricle, both of which are medi[illegible]. Destructive counterparts include familial heart bloc[illegible]tatal arrhythmias, similar results in the long QT syndr[illegible]hogenesis of both Uhl's anomaly and arrhythmogenic right ventricu[illegible]a; in each apoptosis is an important factor. With permission, I[illegible]al Review of Physiology Volume 60, 1998, by Annual Revie[illegible]p://www.annurev.org).

TEXT:

KEY WORDS: sinus node, AV node, right ventricular involution, Uhl's anomaly

INTRODUCTION

Although apoptosis remains an unfamiliar word and concept for some

physicians and scientists, an understanding of apoptosis has already become essential in explaining many fundamental biological processes such as those by which a variety of cardiovascular functions and diseases emerge and evolve. The word apoptosis was introduced by Kerr, Wyllie & Currie (1) to explain a generally ignored form of cell death very different from necrosis. It is an original Greek word referring to a quiet dropping out process similar to leaves falling from a tree, an image resembling most histologic examples of apoptosis. An excellent history of earlier morphological descriptions of this type over the past century has recently been published by Majno & Joris (2), but the broad significance of apoptosis to explain many normal biological functions, such as morphogenesis and the pathogenesis of a variety of immunological and malignant diseases, only began to be recognized more widely through several perceptive interpretations by Wyllie and his colleagues (3-5).

Regrettable delay in the appreciation of major opportunities for understanding the pathogenesis of disease is nothing new. For example, the pioneering reports of Glucksmann (6), Saunders (7), and Pexeider (8), in which they emphasized that cell death was not only normal but an essential component of morphogenesis, did not receive the attention of scientists or physicians that those reports warranted. Familiar examples of useful morphogenesis include the crucial role of cell death (by apoptosis) in the fetal transformation of interdigital webs into fingers and toes, and the postnatal death of enormous numbers of neurons in the development of useful signaling pathways during normal maturation of the brain.

When I first suggested that cell death was ubiquitous and also normal in the postnatal maturation of the AV (atrioventricular) node and His bundle and that it could pose a hazard for crib death (9), given certain concurrent factors present by chance (10), the idea was harshly criticized (11-13) for a variety of stated reasons. Some said that cell death was never normal, some said that they did not see examples of such cell death in the conduction system of the heart, while still others said yes they saw exactly what I described but that it was a normal phenomenon and not one of cell death because "there was no evidence of necrosis." Apoptosis was presumably an unfamiliar or unacceptable phenomenon for these critics. But as recently emphasized in my refutations of those criticisms, cell death is a normal component of postnatal morphogenesis of the human cardiac conduction system, and apoptosis is a major and possibly the principal mechanism by which this occurs (14).

However, the fact that apoptotic cell death may very often be useful does not mean that apoptosis is always a beneficial process. This seeming paradox--in some respects the yin and yang of cardiology--is better understood when one considers the multitude of different promoters or inhibitors of apoptosis, each of which has a progression that in some circumstances may be reversible, but at a later stage becomes irreversible, thus committing the cell inevitably to die. Unsurprisingly, the balance betwen promotion and inhibition of apoptosis often changes with time, or as a consequence of associated metabolic or toxic influences, and it differs in different organs of the mammalian body and differs to some degree between species. Whether apoptosis in the human heart is beneficial or harmful depends significantly upon when it happens and how long it lasts. It is the purpose of this review to examine both beneficial and harmful effects of apoptosis in the human heart and to briefly discuss their clinical significance.

ILLUSTRATIVE EXAMPLES OF NORMAL AND ABNORMAL CONSEQUENCES OF APOPTOSIS IN THE HUMAN HEART

POSTNATAL MORPHOGENESIS OF THE CARDIAC CONDUCTION SYSTEM

Both the sinus node and the AV conduction system (node, His bundle, an
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4/7/8 (Item 2 from file: 98)
DIALOG(R)File 98:General Sci Abs/Full-Text

03546695 H.W. WILSON RECORD NUMBER: BGSI97046695 (THIS IS THE FULLTEXT)
Cellular and molecular mechanisms of pulmonary vascular remodeling.
Stenmark, K. R
Mecham, R. P
Annual Review of Physiology (Annu Rev Physiol) v. 59 ('97) p. 89-144
LANGUAGE: English
COUNTRY OF PUBLICATION: United States
WORD COUNT: 25314

ABSTRACT: In many organs and tissues, the cellular response to injury is associated with a reiteration of specific developmental processes. Studies have shown that, in response to injury, vascular wall cells in adult organisms express genes or gene products characteristic of earlier developmental states. Other genes, expressed preferentially in adult cells in vivo, are down-regulated following injurious stimuli. Complicating matters, however, are recent observations demonstrating that the vascular wall is comprised of phenotypically heterogeneous subpopulations of endothelial cells, smooth muscle cells, and fibroblasts. It is unclear how specific subsets of cells respond to injury and thus contribute to the vascular remodeling that characterizes chronic pulmonary hypertension. This review discusses vascular development in the lung and the cellular responses occurring in pulmonary hypertension; special attention is given to heterogeneity of responses within cell populations and reiteration of developmental processes. With permission, from the Annual Review of Physiology Volume 59, 1997, by Annual Reviews Inc. (http:/www.annurev.org).

TEXT:

KEY WORDS: pulmonary hypertension, lung development, vascular development, vascular injury, smooth muscle cells, fibroblasts, endothelial cells

INTRODUCTION

In several adult tissues (e.g. liver and heart), the cellular response to injury is associated with a reiteration of specific developmental processes. This seems to be true for the vasculature as well. Studies have shown that adult smooth muscle cells (SMCs) responding to injury express genes or gene products (such as tropoelastin, fibronectin, tenascin, F31/H19, cytokeratin 8, and extra domain-A fibronectin) characteristic of earlier developmental states (76, 90, 101, 113, 134, 141). In addition, genes such as smooth muscle-specific a-actin, tropomyosin, desmin, and myosin, which are expressed preferentially in adult cells in vivo, are down-regulated when adult cells are stimulated to migrate and divide following injurious stimuli (116, 117). Similar though less extensive reports exist regarding re-expression of developmentally regulated genes by endothelial cells and fibroblasts in response to injury. Complicating matters, however, are recent observations demonstrating that the vascular

media is comprised of phenotypically heterogeneous subpopulations of SMCs. It is unclear if all adult medial SMC subpopulations are capable of responding to injury with changes in gene expression and replicative potential, or if the post-injury response is limited to specific subsets of cells within the vessel wall (222). The goal of this review is thus twofold: (a) to discuss vascular development in the lung, especially the mechanisms that control growth and differentiation (in an effort to lay the groundwork for understanding the cellular responses and the mechanisms that control them in the setting of pulmonary vascular injury) and (b) to discuss the cellular changes that occur in various forms of pulmonary hypertension, with special attention to heterogeneity of responses within cell populations and reiteration of developmental processes (Figure 1).

DEVELOPMENT OF THE PULMONARY VASCULATURE

The lung is a highly complex organ comprised of more than 40 different cell types (38) that are involved in both respiratory and nonrespiratory functions. Despite its eventual complexity of structure and function, the lung has ostensibly simple beginnings. The lung epithelium originates as paired outpocketings from the floor of the pharyngeal endoderm that expand into mesenchyme derived from splanchnic mesoderm. The epithelial rudiments subsequently undergo a series of repetitive branchings to give rise to the pulmonary tree. Much effort has been directed at defining the mechanisms that regulate normal epithelial pattern formation (termed branching morphogenesis), but much less is known about how the pulmonary vasculature is formed. The development and maintenance of normal vascular structure clearly plays a critical role in lung function, yet several major questions about the regulation of this process in the normal and diseased lung remain unanswered.

ENDOTHELIAL CELL REPLICATION AND VASCULOGENESIS IN THE DEVELOPING LUNG

In the embryonic lung, endothelial precursor cells (angioblasts) initially form a primary vascular plexus within the tissue, which eventually links up to the main circulation, of sixth branchial arch origin, coming from the heart. Studies using intracoelomic chimeric recombinations between quail and chick embryonic lungs have demonstrated that the endoderm seems to control lung vasculogenesis by inducing the emergence of endothelial cells in its associated mesoderm (184). Furthermore, tissue mixing experiments also suggest that vessel formation in the lung, like branching morphogenesis, may depend on the interaction between epithelium and mesenchyme (129, 143, 212). Thus a true reciprocity would exist in which epithelial proliferation and differentiation are induced by an as-yet-unidentified mesenchymal cell population. The induced epithelium (or a subpopulation of cells thereof) in turn produces factors that stimulate endothelial cell proliferation and organization. The endothelium itself may then produce factors to recruit other mesenchymal cells (e.g. SMC precursors) necessary for the completion of vessel structure.

Specific growth factors found in the developing lung, including acidic fibroblast growth factor (aFGF), basic fibroblast growth factor (bFGF), and platelet-derived endothelial cell growth factor (PD-ECGF), regulate endothelial cell proliferation in vivo and in vitro (81, 84, 99, 216). Another candidate molecule likely to play a major role in lung vascular development is vascular endothelial growth factor (VEGF), a member of the PDGF family that is identical to vascular permeability factor (157) and vasculotropin (190). It is abundant in highly vascularized tissues such as kidney, placenta, and lung. Unlike the FGFs, VEGF appears to be an

endothelial-specific mitogen and is involved in both
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4/7/9 (Item 1 from file: 144)
DIALOG(R)File 144:Pascal

13863741 PASCAL No.: 99-0041435
Influence des alleles de l'apolipoproteine E sur la restenose apres angioplastie coronaire chez la femme
(Influence of apolipoprotein E alleles on post-angioplasty **restenosis** in woman)
FLORK L; JOUANEL P; LUSSON J R; LEAUTE S; DAUPHIN C; MOTREFF P; JUSTIN E P; LAMAISON D; BOIRE J Y; CASSAGNES J
Service de cardiologie, CHU G-Montpied, 63009 Clermond-Ferrand, France; Laboratoire de biochimie, CHU Hotel-Dieu, 63009 Clermond-Ferrand, France; ERIM, Faculte de medecine, BP 38, 63001 Clermond-Ferrand, France
Journal: Archives des maladies du coeur et des vaisseaux, 1998, 91 (12) 1475-1479
ISSN: 0003-9683 CODEN: AMCVAN Availability: INIST-887; 354000073231900050
No. of Refs.: 23 ref.
Document Type: P (Serial) ; A (Analytic)
Country of Publication: France
Language: French
Meme si l'emploi d'endoprothese en diminue l'incidence, le phenomene de restenose coronaire apres dilatation percutanee reste entier. L'influence des lipoproteines dans le developpement de l'atherosclerose n'est plus a demontrer, mais leur role dans la restenose est plus discutable. Des resultats contradictoires ont ete publies a propos de la responsabilite du genotype de l'Apo E. Dans un premier travail, nous avons mis en evidence la relation LP (a) et coronaropathie plus etroite chez la femme que chez l'homme, Un sous-groupe de patientes qui ont eu une angioplastie dont le profil lipidique etait bien etabli a ete analyse en se focalisant sur les alleles de l'Apo E. Methodes : 59 patientes ont eu une angioplastie parmi lesquelles 35 monotronculaires, 20 bitronculaires et 4 tritronculaires. Une coronarographie de controle a ete pratiquee chez 40 patientes. Un interrogatoire telephonique a ete realise entre 12 et 22 mois apres la dilatation a l'ensemble de la population. Les apolipoproteines A1, B. LP (a) et LP A1 sont dosees par techniques immunologiques, turbidimetriques ou electro-immunologiques. Le genotypage de l'Apo E est realise a l'aide du kit Inno-Lipa. Resultats : la repartition des patientes est la suivante : 18 restenoses angiographiques (groupe A), 20 coronarographies sans restenose (groupe B), 41 sans restenose angiographique (20) ou clinique (21) (groupe C). Dans le groupe A, la LP (a) depasse largement le seuil de 0,30 g/L. L'**allele** e4 est associe a un cholesterol total et sa fraction LDL les plus eleves. **Il** n'y a pas de difference significative entre le genotype Apo E des differents groupes ni en fonction de la severite des lesions. En conclusion : si l'**allele** e4 est plus frequemment associe a un taux eleve de LDL cholesterol et LP (a), nous n'avons pas prouve son role dans le processus de restenose. Un nombre de malades plus Important est necessaire et des travaux complementaires souhaitables pour comprendre les mecanismes inflammatoires et/ou immunologiques par lesquels l'Apo E pourrait Intervenir dans la restenose.

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4/7/10 (Item 2 from file: 144)
DIALOG(R)File 144:Pascal

13475066 PASCAL No.: 98-0172121
EPIDEMIOLOGIE DE LA MALADIE CORONAIRE : RECHERCHE DES DETERMINANTS GENETIQUES DE LA VASOMOTRICITE CORONAIRE ET DE LA RESTENOSE
(CORONARY DISEASE EPIDEMIOLOGY : CORONARY AND **RESTENOSIS** GENETIC RISK FACTORS DETERMINATION)
AMANT Carole; AMOUYEL Philippe, dir
Universite de Lille 1, Villeneuve-d'Ascq, Francee
Univ.: Universite de Lille 1. Villeneuve-d'Ascq. FRA Degree: Th. doct. 1997-12; 1997 183 p.
Availability: INIST-T 116002; T97LIL10134 0000; RBCCN-590092102; T97LIL10134 0000
No. of Refs.: 214 ref.
Document Type: T (Thesis) ; M (Monographic)
Country of Publication: France
Language: French Summary Language: French; English
L'insuffisance coronaire et l'infarctus du myocarde sont les premieres causes de mortalite et de morbidite dans les pays industrialises. Comme dans la plupart des maladies multifactorielles, de nombreux facteurs de risque predisposent au developpement et a la survenue de la maladie coronaire. **Il** s'agit des facteurs de risque environnementaux et des facteurs de susceptibilite genetique. Dans le travail presente ici, nous nous sommes focalises sur des genes candidats issus du systeme renine-angiotensine. Les etudes developpees ont consiste a mesurer le degre d'implication de ce systeme dans les phenomenes de restenose et de vasoconstriction accrue. Nos resultats suggerent que l'**allele** D du gene de l'Enzyme de Conversion de l'Angiotensine I (ECA) constitue un facteur de risque majeur d'occlusion apres angioplastie conventionnelle et de restenose apres angioplastie avec pose d'endoprothese coronaire, le stent. Nous avons aussi mis en evidence que l'**allele** C du gene du recepteur de l'angiotensine II de type 1 (AT1) est lie a une vasoconstriction coronaire augmentee. Cliniquement, ces resultats sont interessants car ils permettent d'identifier les patients a haut risque de restenose et/ou de vasoconstriction augmentee dans le but de prevenir les accidents coronaires graves qui pourraient survenir, tels qu'un infarctus post-angioplastie ou un vasospasme. Cependant d'autres etudes sont necessaires pour verifier nos resultats tant au niveau epidemiologique qu'aux niveaux clinique et physiologique.

4/7/11 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11567561 21405569 PMID: 11514491
Interleukin 1 receptor antagonist gene **polymorphism** and **restenosis** after coronary angioplasty.
Francis SE; Camp NJ; Burton AJ; Dewberry RM; Gunn J; Stephens-Lloyd A;

Cumberland DC; Gershlick A; Crossman DC
Cardiovascular Medicine Group, Division of Clinical Sciences, Clinical Sciences Centre, University of Sheffield, Northern General Hospital, Sheffield S5 7AU, UK.
Heart (England) (Sep 2001,) 86 (3) p336-40, ISSN 1468-201X
Journal Code: CEN
Languages: ENGLISH
Document type: Journal Article
Record type: In Process
BACKGROUND: Percutaneous transluminal coronary angioplasty (PTCA) is limited by the recurrence of luminal stenosis, which occurs in up to 50% of procedures. It has been shown that patient specific factors, perhaps genes, contribute to this process. OBJECTIVE: To determine whether completion of healing after PTCA is part of an acute self limiting inflammatory process and whether **polymorphism** at important inflammatory gene loci might determine susceptibility to **restenosis** after PTCA. DESIGN: DNA samples were collected from 171 patients attending for elective PTCA in Sheffield (S) and Leicester (L), who were scheduled to undergo follow up angiography (at four months (L) or six months (S)) as part of other **restenosis** studies. At follow up angiography, the patients were separated into restenosers (> 50% luminal narrowing) and non-restenosers (< 50% luminal narrowing). Four DNA polymorphisms within **interleukin** 1 (**IL**-1) related loci (**IL**-1A (-889), **IL**-1B (-511), **IL** -1B (+3954), and **IL** -1RN intron 2 VNTR (variable number tandem repeat)) were genotyped using methods based on polymerase chain reaction. Significance was assessed by chi(2) analysis of the relevant contingency table, and the magnitude of effect was estimated by calculating odds ratios. The Mantel-Haenszel (MH) test was applied to summarise data across the two populations. RESULTS: **Allele** 2 at **IL**-1RN (**IL** -1RN*2) was significantly over represented in the non-restenoser group (L+S, 34% v 23% in restenosers). Furthermore, **IL**-1RN*2 homozygosity was increased in the non-restenoser population compared with the restenosers (MH test: p = 0.0196 (L+S); p = 0.031 (L+S, single vessel disease only), and the effect seemed to be restricted to the single vessel disease subpopulation. For other **polymorphism** within **IL**-1 related loci no significant associations were found with either **restenosis** or non-**restenosis**. CONCLUSIONS: **IL**-1RN*2 may be associated with protection from **restenosis** after PTCA for individuals with single vessel disease. As this **polymorphism** has functional significance, this finding suggests that alteration in an individual's inflammatory predisposition may modulate the blood vessel response to injury.
Record Date Created: 20010821

4/7/12 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

135032750 CA: 135(3)32750s PATENT
Interleukin-1 homologue, MAT IL-1H4
INVENTOR(AUTHOR): Kumar, Sanjay; McDonnell, Peter C.; Young, Peter R.
LOCATION: USA
ASSIGNEE: Smithkline Beecham Corporation
PATENT: PCT International ; WO 0140247 A1 DATE: 20010607
APPLICATION: WO 2000US32521 (20001130) *US 452140 (19991201)
PAGES: 30 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C07H-021/02A; C12N-015/00B; C12N-005/00B; C07K-001/00B; C07K-016/00B

DESIGNATED COUNTRIES: JP DESIGNATED REGIONAL: AT; BE; CH; CY; DE; DK; ES ; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; TR

SECTION:

CA215005 Immunochemistry
CA201XXX Pharmacology
CA203XXX Biochemical Genetics
CA209XXX Biochemical Methods
CA214XXX Mammalian Pathological Biochemistry

IDENTIFIERS: MAT interleukin 1H4 agonist antagonist screening, antibody inflammation mature interleukin 1H4 inhibitor

DESCRIPTORS:

Respiratory distress syndrome...
acute; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Diagnosis...
agents; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Immunoassay...
enzyme-linked immunosorbent assay; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Heart,disease...
failure; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Transplant and Transplantation...
graft-vs.-host reaction; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Intestine,disease...
inflammatory; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Brain,disease...
injury; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

AIDS(disease)... Allergy... Alzheimer's disease... Antibodies... Arthritis ... Asthma... Atherosclerosis... Autoimmune disease... Bone,disease... cDNA sequences... Drug screening... Fusion proteins(chimeric proteins)... Infection... Inflammation... Ischemia... Labels... Lymphoproliferative disorders... Molecular cloning... mRNA... Mutation... Neoplasm... Nucleic acid hybridization... Oligonucleotides... Osteoporosis... Protein sequences ... Psoriasis... Septicemia... Stress,animal... Susceptibility(genetic)...

Transplant rejection...
interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Probes(nucleic acid)...
labeled; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

Artery,disease...
restenosis; interleukin 1H4 and mature IL-1H4 for screening agonists and antagonists for treating inflammation, infection, cancer, autoimmune disease and others

12219937 BIOSIS NO.: 199900514786
Insertion/deletion **polymorphism** of the **angiotensin**-converting enzyme and **restenosis** after coronary stent placement.
AUTHOR: Kastrati Adnan; Koch Werner; Elezi Shpend; Kettling Corinna; Mehilli Julinda; Von Beckerath Nicolas; Schomig Albert
AUTHOR ADDRESS: Dtsch. Herzzent., 1. Med. Klin., TUM, Munich**Germany
JOURNAL: Circulation 98 (17 SUPPL.):pI433 Oct. 27, 1998
CONFERENCE/MEETING: 71st Scientific Sessions of the American Heart Association Dallas, Texas, USA November 8-11, 1998
SPONSOR: The American Heart Association
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English

7/7/19 (Item 19 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12211510 BIOSIS NO.: 199900506359
Not the **ACE** gene **polymorphism**, but the E-selectin gene **polymorphism** helps to predict **restenosis** after coronary angioplasty.
AUTHOR: Rauchhaus Mathias(a); Francis Darrel P; Schmidt Hendrik; Teichmann Wilhelm; Glaeser Christiane
AUTHOR ADDRESS: (a)ICSM, NHLI, London**UK
JOURNAL: Circulation 98 (17 SUPPL.):pI393-I394 Oct. 27, 1998
CONFERENCE/MEETING: 71st Scientific Sessions of the American Heart Association Dallas, Texas, USA November 8-11, 1998
SPONSOR: The American Heart Association
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English

7/7/22 (Item 22 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12192449 BIOSIS NO.: 199900487298
Long-term clinical outcome of coronary stenting: Role of the D **allele** of the **ACE** gene, and importance of the angiographic pattern of **restenosis**.
AUTHOR: Ribichini F(a); Ferrero V(a); Vado A(a); Steffenino G(a); Dellavalle A(a); Russo P(a); Matullo G; Colajanni E; Guarrera S; Piazza A ; Uslenghi E(a)
AUTHOR ADDRESS: (a)Divisione di Cardiologia, Ospedale Santa Croce di Cuneo, Cuneo**Italy
JOURNAL: European Heart Journal 20 (ABSTR. SUPPL.):p602 Aug., 1999
CONFERENCE/MEETING: XXIst Congress of the European Society of Cardiology Barcelona, Spain August 28-September 1, 1999
SPONSOR: European Society of Cardiology
ISSN: 0195-668X
RECORD TYPE: Citation
LANGUAGE: English

7/7/28 (Item 28 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11741680 BIOSIS NO.: 199800522376
Significance of **angiotensin** I-converting enzyme gene **polymorphism** as risk factor for stent **restenosis**.
AUTHOR: Guerlek Adalet(a); Bokesoy Isik; Gulec Sadi(a); Karabult Halil; Toydemir Reha; Aras Omer; Oral Dervis(a)
AUTHOR ADDRESS: (a)Med. Sch. Ankara Univ., Dep. Cardiology, Ankara**Turkey
JOURNAL: European Heart Journal 19 (ABST. SUPPL.):p34 Aug., 1998
CONFERENCE/MEETING: XXth Congress of the European Society of Cardiology Vienna, Austria August 22-26, 1998
SPONSOR: European Society of Cardiology
ISSN: 0195-668X
RECORD TYPE: Citation
LANGUAGE: English

7/7/77 (Item 17 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

06053694 Genuine Article#: XE898 Number of References: 0
Title: **Angiotensin**-converting enzyme gene **polymorphism** and risk of **restenosis** after coronary stenting
Author(s): Barberis P; Merlini PA; Cavalotti C; Bernardi F; Marchetti G; Ferraresi P; Laudisa ML; Ardissino D
Corporate Source: POLICLIN SAN MATTEO,IRCCS, DIV CARDIOL/I-27100 PAVIA//ITALY/; UNIV FERRARA,DEPT BIOCHEM & MOL BIOL/I-44100 FERRARA//ITALY/
Journal: THROMBOSIS AND HAEMOSTASIS, 1997, S (JUN), PP2125-P2125
ISSN: 0340-6245 Publication date: 19970600
Publisher: F K SCHATTAUER VERLAG GMBH, P O BOX 10 45 45, LENZHALDE 3, D-70040 STUTTGART, GERMANY
Language: English Document Type: MEETING ABSTRACT

343779-79-3 unclaimed sequence; interleukin-1 homolog, MAT IL-1H4

4/7/13 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

134026093 CA: 134(3)26093t PATENT
Diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
INVENTOR(AUTHOR): Kornman, Kenneth S.; Duff, Gordon W.; Crossman, David C.; Francis, Sheila E.; Stephenson, Katherine
LOCATION: USA
ASSIGNEE: Interleukin Genetics, Inc.
PATENT: PCT International ; WO 200071753 A2 DATE: 20001130
APPLICATION: WO 2000US14299 (20000524) *US 317674 (19990524) *US 431352 (19991101)
PAGES: 129 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12Q-001/68A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZW; AT ; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA203003 Biochemical Genetics
CA201XXX Pharmacology
CA209XXX Biochemical Methods
CA214XXX Mammalian Pathological Biochemistry
CA215XXX Immunochemistry
IDENTIFIERS: interleukin 1 genotyping restenosis diagnosis therapeutic
DESCRIPTORS:
Nucleic acid hybridization...
allele-specific; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Artery,disease...
coronary, restenosis; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Allele frequency... Anticoagulants... Antihypertensives... Antisense oligonucleotides... Anti-inflammatory agents... DNA sequence analysis... Drug screening... Genetic polymorphism... Genotyping(method)... Hypolipemic agents... Interleukin 1 receptor antagonist... Interleukin 1.alpha.... Interleukin 1.beta.... Interleukin 1... Mutation... Platelet aggregation inhibitors... Primers(nucleic acid)... RFLP(restriction fragment length polymorphism)... Ribozymes... SSCP(single-strand conformation polymorphism) ... Test kits...
diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Diagnosis...
genetic; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Gene,animal...
IL-1RN; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Gene,animal...
IL1A; diagnostics and therapeutics for restenosis based on genotyping

of the interleukin-1 family
Gene,animal...
IL1B; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Heart,disease...
infarction; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Blood vessel,disease...
occlusion; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Genetic methods...
oligonucleotide ligation assay; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Periodontium...
periodontitis; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Nucleic acid amplification(method)...
primer-specific extension; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Artery,disease...
restenosis; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
Oligonucleotides...
triple helix forming; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
CAS REGISTRY NUMBERS:
81295-04-7 81295-06-9 81295-40-1 84522-62-3 86352-30-9 92228-44-9 172306-44-4 diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
140744-82-7 140960-10-7 141002-08-6 nucleotide sequence; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
188135-75-3 188135-76-4 188135-77-5 188135-78-6 188135-79-7 193101-09-6 206073-47-4 224178-70-5 224178-77-2 249263-92-1 249263-94-3 249263-96-5 309983-89-9 309983-90-2 PCR primer; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
244295-37-2 244295-38-3 244295-41-8 244295-42-9 309983-91-3 309983-92-4 309983-93-5 309983-94-6 unclaimed nucleotide sequence; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family

4/7/14 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

134014046 CA: 134(2)14046s PATENT
Diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
INVENTOR(AUTHOR): Francis, Sheila E.; Crossman, David C.; Duff, Gordon W.; Kornman, Kenneth S.
LOCATION: USA
ASSIGNEE: Interleukin Genetics, Inc.
PATENT: PCT International ; WO 200072015 A2 DATE: 20001130
APPLICATION: WO 2000US14775 (20000526) *US 320395 (19990526) *US 431352 (19991101)
PAGES: 122 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: G01N-033/53A

DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZW; AT ; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

SECTION:

CA203003 Biochemical Genetics
CA201XXX Pharmacology
CA209XXX Biochemical Methods
CA214XXX Mammalian Pathological Biochemistry
CA215XXX Immunochemistry

IDENTIFIERS: cardiovascular disease interleukin 1 gene family polymorphism, restenosis interleukin 1 gene family polymorphism, occlusive disorder interleukin 1 gene family polymorphism, fragile plaque disorder interleukin 1 gene family polymorphism, genotyping interleukin 1 gene family cardiovascular disease, RFLP interleukin 1 gene family cardiovascular disease

DESCRIPTORS:

Nucleic acid hybridization...
    allele-specific; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Proteins,specific or class...
    C-reactive, IL-1 genotypes correlated with; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Artery,disease...
    coronary, restenosis; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
DNA sequence analysis... Drug screening... Genotyping(method)... Interleukin 1 receptor antagonist... Interleukin 1.alpha.... Interleukin 1.beta.... Interleukin 1... Mutation... Primers(nucleic acid)... RFLP(restriction fragment length polymorphism)... SSCP(single-strand conformation polymorphism)... Susceptibility(genetic)... Test kits...
    diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Cardiovascular system...
    disease; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Blood vessel,disease...
    fragile plaque; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Diagnosis...
    genetic; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Gene,animal...
    IL-1RN; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Gene,animal...
    IL1A; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Gene,animal...
    IL1B; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Heart,disease...
    infarction; diagnostics and therapeutics for cardiovascular disorders

based on genotyping of the interleukin-1 family
Lipoproteins...
low-d., IL-1 genotypes correlated with; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Lipoproteins...
Lp(a), IL-1 genotypes correlated with; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Angiogenesis...
neovascularization, IL-1 genotypes correlated with; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Blood vessel,disease...
occlusion; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Genetic methods...
oligonucleotide ligation assay; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Periodontium...
periodontitis; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Nucleic acid amplification(method)...
primer-specific extension; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Artery,disease...
restenosis; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
Brain,disease...
stroke; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
CAS REGISTRY NUMBERS:
57-88-5 biological studies, IL-1 genotypes correlated with; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
81295-04-7 81295-06-9 81295-40-1 81811-56-5 84522-62-3 86352-30-9 92228-44-9 diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
188135-76-4 188135-77-5 188135-78-6 188135-79-7 193101-09-6 193101-10-9 224178-77-2 249263-92-1 249263-94-3 249263-96-5 PCR primer; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
244295-37-2 244295-38-3 244295-41-8 244295-42-9 309983-91-3 309983-92-4 309983-93-5 309983-94-6 unclaimed nucleotide sequence; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family

4/7/15 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

133262303 CA: 133(19)262303m PATENT
Human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
INVENTOR(AUTHOR): Hayden, Michael R.; Wilson, Angela R.; Pimstone, Simon

N.
LOCATION: Can.,
ASSIGNEE: University of British Columbia; Xenon Bioresearch, Inc.
PATENT: PCT International ; WO 200055318 A2 DATE: 20000921
APPLICATION: WO 2000IB532 (20000315) *US PV124702 (19990315) *US PV138048 (19990608) *US PV139600 (19990617) *US PV151977 (19990901)
PAGES: 229 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-015/12A; C07K-014/705B; C12N-005/10B; A01K-067/027B; C12N-015/00B; A61K-038/17B; A61K-048/00B; A61K-038/45B; A61K-031/00B; A61K-031/70B; G01N-033/68B; C12Q-001/68B; C12N-015/11B DESIGNATED COUNTRIES: AE; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SL; SZ ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA203003 Biochemical Genetics
CA201XXX Pharmacology
CA206XXX General Biochemistry
CA213XXX Mammalian Biochemistry
IDENTIFIERS: sequence human gene ABC1 transporter cDNA, cholesterol transport familial HDL deficiency ABC1 gene mutation
DESCRIPTORS:
Transport proteins...
ABC1 (ATP binding cassette-contg. 1); human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Genetic vectors...
ABC1 gene-contg.; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
RNA splicing...
ABC1 mutation affecting, disease and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Interleukin 1.beta....
ABC1 transport of; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Gene,animal...
ABC1; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Chicken(Gallus domesticus)... Mammal(Mammalia)...
ABC1/ABC1 mutant-expressing; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Brain,disease...
cerebrovascular; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Artery,disease...
coronary, restenosis; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Artery,disease...
coronary; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Mutation...
deletion, of ABC1 gene, disease and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Cardiovascular system...
disease; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

Disease,animal...
familial HDL deficiency, ABC1 gene mutations and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
cDNA sequences...
for human ABC1 transporter
Mutation...
frameshift, of ABC1 gene, disease and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Lipoproteins...
high-d., cholesterol; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Alzheimer's disease... Neoplasm... Niemann-Pick disease...
human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Chromosome...
human 9, ABC1 gene mapped to; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Nervous system...
Huntington's chorea; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Mutation...
nonsense, of ABC1 gene, disease and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Genetic mapping... Genetic polymorphism... mRNA... Promoter(genetic element)... RFLP(restriction fragment length polymorphism)...
of ABC1 gene; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Biological transport...
of cholesterol; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Protein sequences...
of human ABC1 transporter
DNA sequences...
of human ABC1 transporter gene ABC1 exons and flanks
Blood vessel,disease...
peripheral; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Mutation...
substitution, of ABC1 gene, disease and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Disease,animal...
Tangier, ABC1 gene mutations and; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Antidiabetic agents...
thiozolidinediones, modulation of ABC1 with; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
Brain,disease...
X-linked adrenoleukodystrophy; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
CAS REGISTRY NUMBERS:
296343-83-4 296343-89-0 296787-98-9 amino acid sequence; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
56-65-5 biological studies, binding/hydrolysis by ABC1 of; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease

57-88-5 biological studies, human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
50-28-2 59-67-6 biological studies, modulation of ABC1 with; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
943-45-3D derivs., inhibitors, modulation of ABC1 with; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
506-32-1D derivs., modulation of ABC1 with; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
9028-35-7 71160-24-2 inhibitors, modulation of ABC1 with; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease
18104-45-5 28822-58-4 37353-31-4 50892-23-4 78111-17-8 98462-03-4 98524-19-7 141436-78-4 142008-29-5 modulation of ABC1 with; human ABC1 transporter and DNA and methods for modulating cholesterol levels and diagnosing disease